

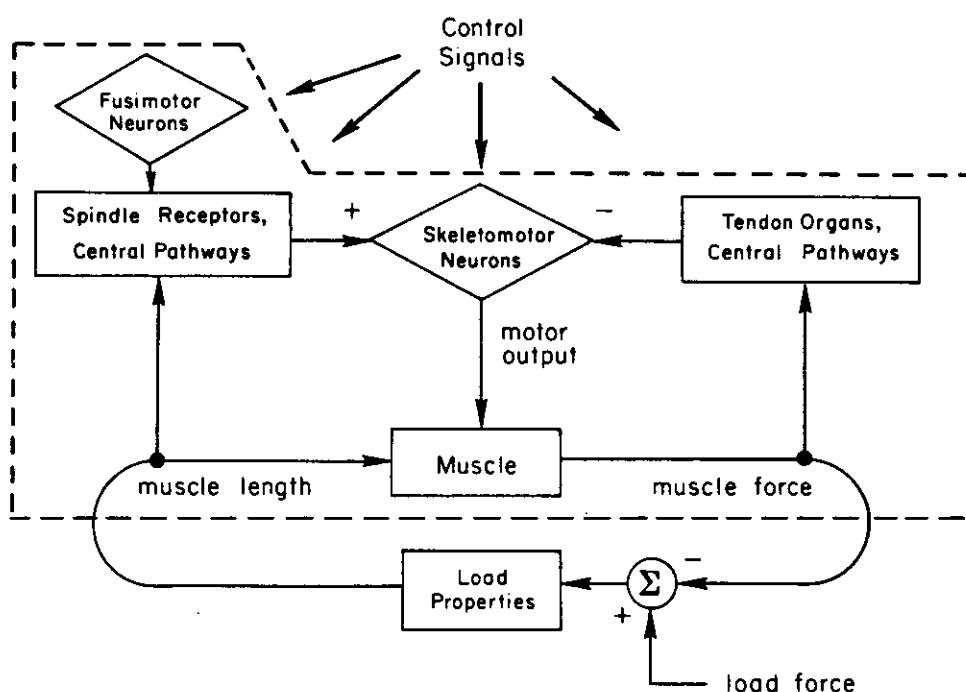
# Neural control of muscle length and tension

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**FIG. 1.** Basic organizational plan of the motor servo. Muscle and load forces act on load properties (e.g., inertia) to produce length changes. Muscle force is regulated by motor output from skeletomotor neurons (reflex action) but also varies in response to changes in length (so-called mechanical response). Muscle length (and velocity) is monitored by spindle receptors and force by Golgi tendon organs. These signals provide excitation and inhibition, respectively, to skeletomotor neurons by way of central pathways (certainly segmental, and perhaps also suprasegmental). Neural control signals are sent to skeletomotor and fusimotor neurons and to interneurons in the reflex pathways.

(and its derivatives) sensed by muscle spindle receptors, and muscle force or tension sensed by Golgi tendon organs.

The motor servo is a particularly important topic in motor control, since it is involved, in one way or another, in all motor acts, whether these are simple responses to muscle stretch or complex movements produced by central motor commands. In effect, the motor servo functions as a *final common processor* of motor commands that are sent to it from other regions of the nervous system in ensembles of nerve fibers. This role is analogous, though on another hierarchical level, to the role of the motor unit, which serves as the final common pathway for motor integration at the cellular level. A productive approach to the understanding of how the motor servo translates central motor commands into changes in muscle length and force has been to study how these variables are controlled when mechanical disturbances, rather than central commands, act as inputs. Furthermore these responses to disturbances in mechanical load are intrinsically important themselves, since they represent the first line of defense, so to speak, in the continual interaction that occurs between an organism and its mechanical environment.

The operations of the motor servo have been the subject of extensive experimental investigation throughout the fifty some years since the stretch reflex was first subjected to detailed analysis by Liddell and

Sherrington (170). Frequent attempts have been made to incorporate the empirical findings into the conceptual framework of feedback-control theory, and the hypotheses thus generated have helped to direct attention to important unresolved questions requiring further experimentation. This chapter lays out the major theoretical ideas and then reviews the experimental evidence regarding these hypotheses. In addition we include background sections on control theory, muscle mechanics, central pathways, and simplified animal preparations. The latter are arranged so that readers with experience in any of these particular topics can easily skim over the material.

#### CONTROL THEORY CONCEPTS

##### *Systems and Models*

A system can be defined as any assemblage of objects united by processes of physical interaction that relate measurable quantities one to another. The measurements of the relevant quantities are usually expressed as sets of values called signals, variables, or time functions. System theory deals primarily with the relations between such variables, rather than with the actual physical nature of the quantities they describe. One comes to view some of the variables as being determined independently, by causes extrinsic to the system, and others as being dependent on these,

although this distinction is sometimes arbitrary. The independent variables are called inputs, the dependent ones are called states, and the outputs are variables that derive from the states. The system is defined to include all the processes that intervene to relate outputs to inputs. In this section only elementary aspects of system theory are summarized, and the reader is referred to the chapter by Arbib in this *Handbook* for a discussion of more advanced concepts.

A key concept in system theory is the notion of an *operator* or *transfer function*. These equivalent terms arise from an abstraction—a view of a system as a process that operates upon input variables to transform them into output variables. This notion provides a convenient way of thinking about a mathematical model of a system. The model is a set of rules such as an equation or graph that details the steps in the conversion process. Inputs are added, subtracted, multiplied, divided, integrated, and differentiated to convert them into outputs. The equation specifies which of these elementary mathematical operations are required, together with the particular order in which they must be performed. We are all accustomed to using the symbols  $+$ ,  $-$ ,  $\times$ ,  $/$ ,  $\int ( ) dt$ , and  $d( )/dt$  to represent these elementary operations; they are examples of elementary operators.

In other cases it is more convenient to write the equation characterizing a system in a more abstract manner. Then a single symbol, also called an operator, is used to represent a whole sequence of elementary mathematical operations. For example, a general equation characterizing any system is

$$y(t) = S[x(t)] \quad (1)$$

where  $x(t)$ , hereafter written simply as  $x$ , is the input,  $S$  is the system operator, and  $y$  is the output. If the system has more than one input,  $x$  will be a vector consisting of a set of input functions ( $x_1, x_2, \dots, x_n$ ) and, similarly, the output  $y$  may also be a vector. Operator notation obviously saves space in writing equations, but more importantly it encourages abstract thinking about a system as being characterized by a single complex operation. As will be shown shortly, this abstract approach is particularly useful in demonstrating general properties of classes of systems, i.e., conclusions based on very few assumptions regarding the detailed characteristics of the component processes.

Another useful concept is that of inverse operations. The inverse of an operation  $S$ , written  $S^{-1}$ , is an operation that undoes the original one. Thus if  $S$  describes the conversion of an input  $x$  into an output  $y$  (Fig. 2A),  $S^{-1}$  describes a process that converts  $y$  back into  $x$  (Fig. 2B). As a simple example, division by a constant is an operation that is the inverse of multiplication by the same constant. On the other hand, inversion of an operation may be much more complex than this, and in some cases a unique inverse does not exist (a system that squares the input has no unique inverse), or it may exist but yet be impossible to

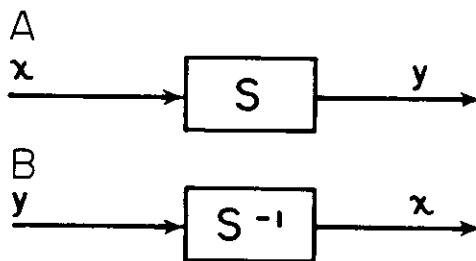


FIG. 2. System models and their inverses. A: the system operator  $S$  converts an input  $x$  into an output  $y$ . B: the inverse of  $S$ , written  $S^{-1}$ , converts  $y$  back into  $x$ . [Adapted from Houk (131). Systems and models. In: *Medical Physiology* (14th ed.), edited by V. B. Mountcastle. St. Louis, MO: The C. V. Mosby Co., 1980.]

construct, in which case it is called unrealizable (the inverse of a pure time delay, a predictor, is unrealizable, since it defies causality).

*Models* are hypotheses describing how a system actually functions. They can be expressed as equations, graphs, block diagrams, physical analogues, or simply as words. The advantage of using mathematics is that this mode of expression is both precise and concise. The disadvantage is that one generally must make simplifying assumptions in order to express the measured properties of a system in the form of equations, and further assumptions may be required to solve the equations. For example, ordinary linear differential equations with constant coefficients, or Laplace transforms of this class of equations, are often used to express models of systems. This is because general techniques for solving these equations are known and relatively easy to apply (131, 212, 215, 251). The use of these equations, however, involves the assumption that the salient properties of a system are captured by a compartmentalized (rather than distributed) model that is linear and has parameters that do not change with time. Alternatively one can explore models that include these complexities by using computer simulation techniques, and there are also some nice mathematical methods for studying the asymptotic behavior of nonlinear systems.

#### Control Systems

Figure 3 introduces a conventional view of the control problem and provides definitions of terms used in this chapter. The purpose of the overall system is to control the values of certain variables within prescribed limits. These controlled variables are a subset of the outputs of what is called the controlled system. An interacting set of physicochemical processes typically forms a controlled system, which is subject to the influences of two categories of input—disturbances and forcing functions. Disturbances are uncontrolled inputs that cause undesired changes of the state of the controlled system, and, hence, of controlled variables. Forcing functions are inputs that can be manipulated to cause (or force) desired changes in state. The function of the controller is to generate appropriate forcing

functions, based on its inputs, called references, set points, or command signals that designate the desired behavior of the overall system.

Another category of input to the controller, not shown in Figure 3, derives from sensors that monitor the states of the controlled system and other variables. Information from sensors is required when disturbances are sufficiently large to produce unacceptable changes in controlled variables. Control systems designed primarily to compensate for these disturbances are called regulators, and the controlled variables may then be called regulated variables.

#### *Feedback, Feedforward, and Adaptive Systems*

The regulatory sensors can be situated to detect potential disturbances or to detect the effects the disturbances have on regulated variables (Fig. 4). The former configuration is called feedforward and the latter is called feedback. Each of these two control strategies has particular advantages and disadvantages.

Because responses of most controlled systems are not instantaneous, disturbances generally occur in ad-

vance of the disturbing effects they provoke. Thus the detection of disturbances by the sensors in a feedforward regulator provides predictive information about impending changes in regulated variables. The controller must then use this information to calculate the effects that measured disturbances are likely to have on the regulated variables and the forcing functions required to counteract these effects. To do this, the controller of a feedforward regulator must, in essence, contain a model of how the controlled system behaves. The precision of regulation clearly depends upon the accuracy of this model. Since the body's controlled systems are frequently nonlinear and time-varying, the models contained within the brain must be either highly complex or else simplified representations of these complex systems. The requirement of complex models for precise regulation is one of the major disadvantages of feedforward control. Another disadvantage derives from the fact that many variables must be monitored to specify all of the potential disturbances and to provide an up-to-date model of a controlled system, since its properties usually change with time. Sensors capable of monitoring all of these variables may not be available, in which case a feedforward regulator is bound to make errors.

One of the intriguing examples of feedforward regulation in body homeostasis concerns the classic conditioned reflex. Pavlov (235), who was one of the first to investigate this phenomenon, demonstrated that practically any sensory cue (the conditioned stimulus) can trigger a given physiological response, provided the former is presented in association with a stimulus (the unconditioned stimulus) that normally elicits the latter. Thus the controller for salivary secretion responds to the sounding of a bell after a period of training during which bell ringing is followed by the introduction of food into the mouth, the latter being a normal stimulus for salivation. In this example the

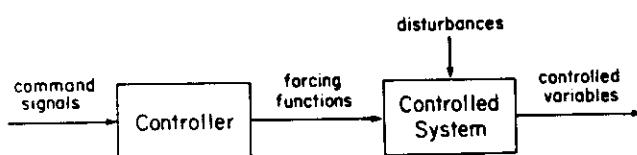


FIG. 3. Basic signals and components of a control system. Command signals designating desired performance are operated on by controller to produce forcing functions. These are inputs to the controlled system that produce desired changes in controlled variables. Uncontrolled inputs act as disturbances and produce undesired changes in controlled variables. [From Houk (131). Homeostasis and control principles. In: *Medical Physiology* (14th ed.), edited by V. B. Mountcastle. St. Louis, MO: The C. V. Mosby Co., 1980.]

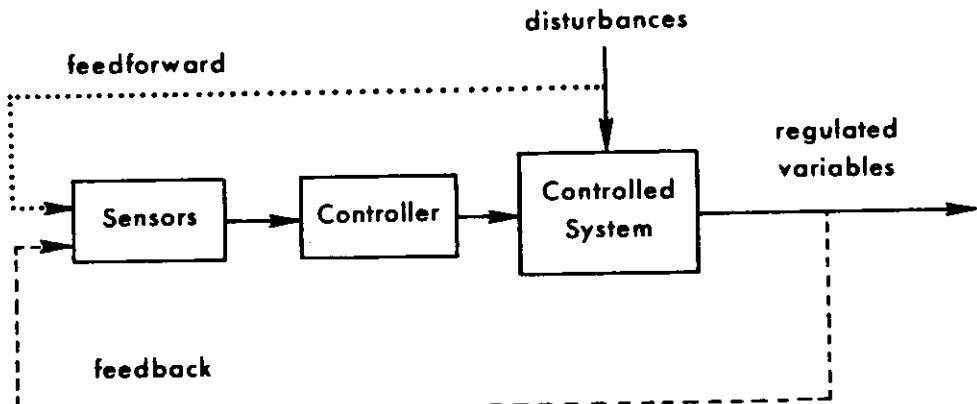


FIG. 4. Feedforward and feedback configurations for regulation. The function of a regulator is to diminish the effects that disturbances have on regulated variables. In a feedforward system regulatory actions are based on signals from sensors that detect potential disturbances, whereas in a feedback system regulatory actions are based on signals from sensors that detect the effects disturbances have on regulated variables. [From Houk (131). Homeostasis and control principles. In: *Medical Physiology* (14th ed.), edited by V. B. Mountcastle. St. Louis, MO: The C. V. Mosby Co., 1980.]

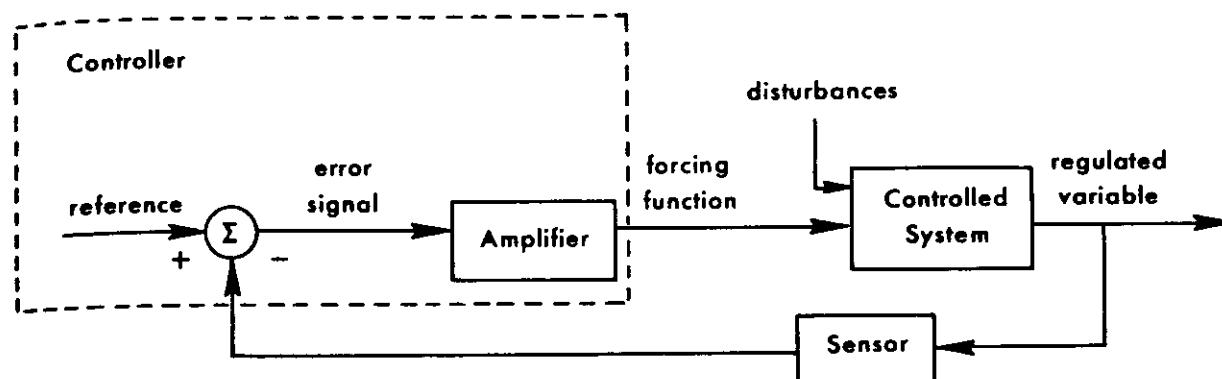


FIG. 5. Simple example of a negative-feedback system. Appropriate forcing functions are generated by 2 elementary operations—error detection (subtraction) and amplification. [From Houk (131). Homeostasis and control principles. In: *Medical Physiology* (14th ed.), edited by V. B. Mountcastle. St. Louis, MO: The C. V. Mosby Co., 1980.]

conditioned reflex provides a feedforward mechanism that prepares the mouth for food that is about to arrive. As is typical of feedforward systems, this mechanism is subject to errors in performance. The first time the bell is not followed by the presentation of food salivation occurs anyway, even though it is inappropriate.

Regulation based on the feedback configuration is free of the major disadvantages of feedforward control, but it has other disadvantages. The fact that feedback control is based on the current and past values of regulated variables eliminates the possibility of completely erroneous regulatory actions. Furthermore the variables that must be monitored are only the regulated ones, and the calculations performed by a feedback controller can be made extremely simple. There are three major disadvantages of feedback regulation: 1) speed—the regulatory actions must await the consequences of the disturbances; 2) stability—due to the presence of a closed loop of control, excessive corrective action can be propagated around the loop in an unending cycle of oscillation; 3) error—the correction depends on the existence of an error and therefore is incomplete (except in special cases in which a perfect integrator is present in the control pathway).

While feedback and feedforward are the two basic strategies for controlling moment-to-moment regulatory actions, a third strategy, adaptive control, functions over longer time periods. Adaptive control is loosely defined as any control that changes to meet changing needs. All regulatory systems satisfy this definition. In the strict sense of the term, however, an adaptive modification is distinguished as a beneficial change in the moment-to-moment properties of a system that occurs over a long time period, one longer than that required for individual responses (3, 7). Biological examples include the hypertrophy of a muscle that occurs as a long-term response to physical training, the acquisition of a new conditioned reflex, or the learning of a new and better response through operant reinforcement.

An adaptive control system has, in addition to the component processes already described, a special subsystem that evaluates the quality of the responses to stimuli, either as they occur naturally or as they are evoked by internally generated test signals. This measure of quality is then used as a basis for adjusting the parameters or structure of the main regulatory system. Thus a well-designed adaptive system should continue to improve its performance based on past experience and readily adjust to new situations. For this reason adaptive control is equated with learning. Needless to say, the general theory of adaptive systems is presently at a rather primitive stage, although definite progress is being made (18, 216, 276, 277).

#### *Principle of Negative Feedback*

The essential feature of a negative-feedback system is the provision for a closed loop of control through which any disturbance in output is opposed. Figure 5 shows how this can be accomplished with the use of a few simple components. A sensor is situated so as to detect one of the outputs of the controlled system, the regulated variable. The output of the sensor, which is proportional to the actual value of the regulated variable, is subtracted from a reference signal, or set point, which represents a desired value of the regulated variable, to form an error signal. This step is called error detection. The error signal is then amplified and sent as a forcing function to the controlled system, thereby forcing the regulated variable back toward the reference value. For example, a disturbance that depresses slightly the value of the regulated variable will result in a small, positive error signal that, when amplified and delivered to the controlled system, will act to elevate (and hence restore) the value of the regulated variable.

The extent to which negative feedback reduces the errors caused by disturbances is uniquely determined by a single parameter (or function) called the loop gain of the system. The meaning of loop gain can be

understood if one considers the output of a general type of feedback regulator to consist of two components as shown in Figure 6: 1) a disturbance component  $y_d$  that represents the uncompensated response to a disturbance, obtained when feedback is absent and 2) a compensatory component  $y_c$  that represents the portion of the output that is attributable to feedback. Loop gain  $G$  can then be defined as the relationship of the compensatory component to the net response  $y$

$$y_c = G[y] \quad (2)$$

$G$  is a composite operation that includes the gain and reference of the controller, the gain of the controlled system, and the sensitivity of the sensor, all lumped together. The loop gain may be a constant (in a linear, static system), but in general it includes lags and delays and therefore is a function of frequency (dynamic system). It may also be a function of amplitude (nonlinear system) and time (time-varying system).

The precise relation between error reduction and loop gain is easily derived as follows

$$y = y_d - y_c \quad (3)$$

$$y = y_d - G[y] \quad (4)$$

$$(1 + G)[y] = y_d \quad (5)$$

The quantity  $(1 + G)$  is itself an operator, and if its inverse exists, the attenuation operator  $A$  can be defined; no assumption of linearity is required

$$y = A[y_d] \quad (6)$$

where

$$A = (1 + G)^{-1} \quad (7)$$

The significance of this result is the following. The variable  $y_d$  represents the severity of a disturbance in terms of the alteration in the controlled variable it would provoke without feedback. Any response to a disturbance, and hence to  $y_d$ , can be considered an error. The operator  $A$  defines the extent to which feedback diminishes this error; error reduction thus depends in an inverse manner on the magnitude of the loop gain. What constitutes good attenuation is of

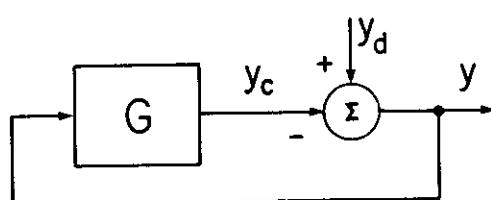


FIG. 6. Generalized negative-feedback system. The regulated variable  $y$  is expressed in terms of 2 components. Open loop (uncompensated) response to a disturbance ( $y_d$ ) and the compensatory response ( $y_c$ ) produced by the feedback system. The system is characterized by loop gain ( $G$ ). [From Houk (131). Homeostasis and control principles. In: *Medical Physiology* (14th ed.), edited by V. B. Mountcastle. St. Louis, MO: The C. V. Mosby Co., 1980.]

course relative, but a loop gain of 9 yields a 10:1 reduction in error [ $A = 1/(1 + 9)$ ]. Negative values for  $G$  represent positive feedback, which increases the error rather than decreasing it.

### Regulated Variables and Properties

It is important to identify regulated variables, since this is one way of specifying the function of a biological feedback system. For cases in which only a single variable is sensed and fed back, it follows that this variable (or a variable derived directly from it) is the regulated one, since the best a negative-feedback system can do is minimize disturbances in the monitored variable. Physiological regulators, however, often receive feedback from several different types of sensors, each of which may monitor a different variable depending on the site of the sensor and its special properties. The latter situation is germane to the analysis of motor servo function, since several categories of muscle receptor serve as feedback sensors, and these receptors are responsive to different mechanical variables.

Given that more than one variable is available as feedback to a controller, the next question is how this additional information is used (cf. ref 131). The possibilities vary along a continuum between two extremes in strategy. One extreme is to use the information to control each of the monitored variables independently and simultaneously. The other is to combine the information about several variables to form a single regulated property. A first step in distinguishing between the alternative strategies is to determine the number of degrees of freedom available in the control of the system. If there is but a single degree of freedom in this control, there cannot be more than one regulated property. As the number of degrees of freedom increases, so does the potential for the simultaneous control of several variables or properties.

### Control Configurations

A servomechanism, or servo for short, is a control system that operates on the principle of negative feedback. The difference between a regulator that uses negative feedback (also called a servoregulator) and a servomechanism is that the reference signal (cf. Fig. 5), previously assumed constant, is allowed to vary as a function of time and is often called a command signal. A derivation similar to that given in *Principle of Negative Feedback*, p. 261, can be used to demonstrate that the controlled variable  $y$  will respond to the command signal  $w$  according to

$$y = SC(1 + FSC)^{-1}[w] \quad (8)$$

where  $S$ ,  $C$ , and  $F$  are operators representing the respective properties of the controlled system, controller, and feedback sensor. Equation 8 does not depend on linearity, but only on the assumption that the

inverse  $(1 + FSC)^{-1}$  exists. Assuming a faithful sensor (with properties  $F = 1$ ) and a high loop gain ( $FSC \gg 1$ ), Equation 8 can be approximated by

$$y \approx SC(SC)^{-1} [w] = w \quad (9)$$

The result  $y \approx w$  means that the system output accurately reproduces the command signal, a condition representing ideal control. Accordingly ideal control can be approached if it is possible to raise the loop gain to very high values. Note that high values of loop gain also make system performance insensitive to nonlinearities and time variations in the properties of the controlled system.

In practice, the extent to which these desirable features can be achieved is limited by the fact that high gains can cause severe stability problems, particularly in loops with time delays. This certainly is a relevant limitation in visually guided tracking movements, due to the long latency in the control pathway associated with a reaction time. It is also a limitation in motor servo performance, and questions concerning stability in motor servo loops are discussed at length in chapters by Rack and by Stein and Lee in this *Handbook*.

*Model-reference* control systems are of several types, but all have in common the use of a component that represents a model of the actual controlled system (cf. refs. 127, 274). The forcing function is sent to both the models  $S_2$  and the actual system  $S_1$ , as shown in Figure 7A, and a special type of error signal, called a reference error, is created by subtracting the actual output from the model output ( $y_2 - y_1$ ). The reference error signal may then be put to different uses as shown by options 1, 2, and 3 in Figure 7A. The basic idea of

model reference comparison was originated by Helmholtz to explain the absence of a sensation of world movement during voluntary eye movement and later applied to a variety of biological problems by Von Holst and Mittelstaedt (285), MacKay and others (cf. 182; the chapter by McCloskey in this *Handbook*). Some of the engineering designs based on this principle are discussed in the following paragraphs.

In a *conditional-feedback* system, option 1, the error is processed (e.g., amplified) and summed with the original forcing function to provide a modified forcing function that is sent to the controlled system (166). This configuration is designed to cancel feedback under conditions in which the controlled system responds precisely as does the model, which is made to represent ideal performance. When disturbances interfere with ideal performance, an error signal is produced, amplified, and applied to the controlled system so as to reduce the error. The efficacy of error reduction depends on the loop gain of the negative-feedback pathway in the same manner discussed previously, and the system is also subject to instability if there is too much delay in the loop or if the gain is too high. The major engineering advantage of the conditional-feedback configuration is that feedforward and feedback features can be designed separately, the former by altering the dynamic properties of the controller (attempting to make  $C$  approach  $S_1^{-1}$ ) and the latter by altering gain and dynamic properties of the feedback loop. This might be advantageous in biological design (evolution) as well.

Additional theoretical advantages result if the reference error is also fed back to the model system (option 2 in Fig. 7A). This creates a positive-feedback

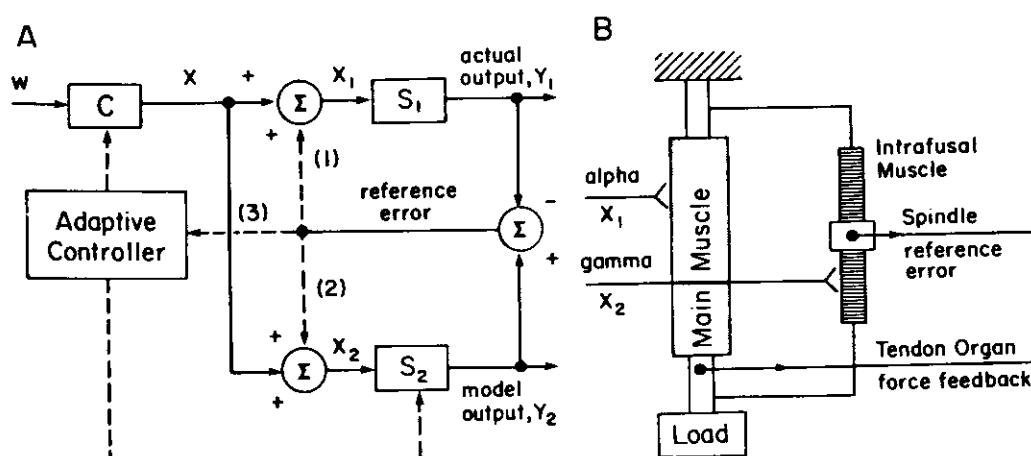


FIG. 7. Model-reference control systems. A: forcing functions are sent to both the controlled system  $S_1$  and a model of the controlled system  $S_2$ . A reference error is computed as the difference between actual output  $y_1$  and model output  $y_2$ . Options 1, 2, and 3 show different ways in which the reference error signal can be used. B: diagram to illustrate how the spindle receptor can be seen as a model reference error detector. The controlled system is main muscle and its load, intrafusal muscle is assumed to the model system, and the spindle afferent endings are situated to detect differences between main and intrafusal muscle shortening. [From Houk (131). Homeostasis and control principles. In: *Medical Physiology* (14th ed.), edited by V. B. Mountcastle. St. Louis, MO: The C. V. Mosby Co., 1980.]

loop, which, if appropriately tuned, can actually result in improved stability along with greater attenuation of errors. In fact errors may be reduced to zero if the gain of the positive-feedback loop is precisely 1, and this gives rise to what has been called a zero-sensitivity system, since it is insensitive to disturbances (274). The third option in Figure 7A involves an adaptive controller that uses the reference error as a basis for modifying the properties of the main controller C and/or the model  $S_2$ .

### Summary

The basic terminology of control theory was introduced and applied to a discussion of the relative merits of several different types of control system. In the following section these ideas are applied to several models of motor servo function.

## HYPOTHESES OF MOTOR SERVO FUNCTION

### Salient Features of the Available Sensors

Since functional characteristics of feedback-control systems are to a considerable extent determined by the properties of its sensors (cf. *Regulated Variables and Properties*, p. 262), it is important to review briefly the salient features of the principle muscle stretch receptors before considering theories of motor servo function.

The specialized efferent innervation of the muscle spindle that was described in the chapter by Matthews in this *Handbook* has been the focus of several of the hypotheses that are described here. These ideas are based on the well-documented dual responsiveness of primary and secondary spindle receptors to muscle stretch and to contraction of the miniature intrafusal muscle fibers upon which the sensory endings lie (cf. Fig. 7B and Fig. 8). Since the central sensory zone of an intrafusal muscle fiber displays less contractile activity than do the poles, it is stretched when the

fiber is activated. This stretch causes receptor discharge rate to rise, as is indicated in Figure 8 by the positive sign at the summing junction. Furthermore the whole structure lies functionally in parallel with the main (or extrafusal) muscle and, as a consequence, when the main muscle lengthens, the intrafusal fiber is also lengthened, again causing stretch of the sensory zone and increased discharge. Conversely, extrafusal shortening causes decreased afferent discharge, which is indicated in Figure 8 by the negative sign at the summing junction. In effect, the spindle receptor responds to the difference between the amounts of intrafusal and extrafusal shortening, where intrafusal length actually refers only to the contractile poles of the intrafusal fiber. The additional influence of  $\gamma$ -dynamic inputs in modifying the sensitivity of the receptor to stretch is shown as a potential adaptive control in Figure 8.

Both primary and secondary endings in muscle spindles show static responses that are approximately proportional to the amount of stretch; thus they are both length sensors. In addition, primary endings show a marked dynamic responsiveness that bears a superficial resemblance to velocity sensitivity, although the actual dependence of response on velocity is rather weak (137).

Golgi tendon organs are selectively sensitive to the force of contraction, more or less independently of the length of the muscle. They do not have high thresholds for discharge as was formerly believed, but instead are sensitive to force variations throughout most of the physiological range (cf. ref. 133).

### Follow-up Servo Hypothesis

One of the first detailed theories concerning the function of the fusimotor system is the follow-up servo hypothesis proposed by Merton in 1951 (210) and elaborated more fully in his 1953 paper (211). Merton postulated that most movements are initiated by motor commands sent to  $\gamma$ -motoneurons, or fusimotor neurons ( $\gamma$ -command in Fig. 8), rather than directly via commands to  $\alpha$ -motoneurons ( $\alpha$ -command in Fig. 8). According to this hypothesis, the commands first travel to the periphery where they produce shortening of the polar zones of intrafusal muscle fibers. The resultant increase in spindle discharge is then conducted back to the spinal cord where it excites the  $\alpha$ -motoneurons (or skeleto-motor neurons) reflexly, leading to activation of the main muscle followed by extrafusal shortening.

The second major postulate in Merton's hypothesis is that the autogenetic reflex pathways function as a servomechanism that regulates muscle length. This is shown in Figure 8 by the negative-feedback loop from spindle receptors through  $\alpha$ -motoneurons to the controlled system consisting of extrafusal muscle and the mechanical load upon which the muscle acts. Note that the only autogenetic reflex pathway included in this hypothesis is the one from spindle receptors.

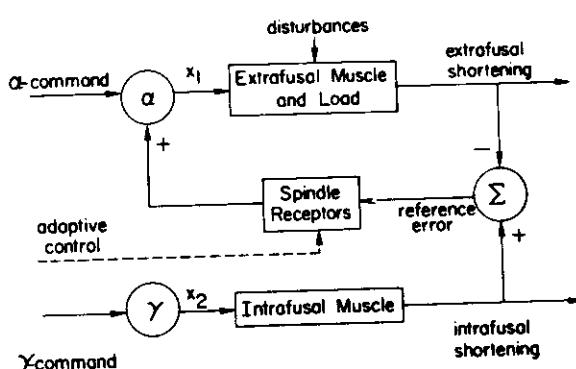


FIG. 8. Block diagram illustrating  $\alpha$ - $\gamma$ -relations.  $\alpha$ -Commands produce shortening of extrafusal muscle and  $\gamma$ -commands produce shortening of intrafusal muscle.  $\alpha$ -Commands reduce spindle discharge, whereas  $\gamma$ -commands increase it, suggesting that spindle receptors may function as reference error detectors as in Figure 7.

Merton assumed that autogenetic inhibition via tendon organs has a high threshold, serving only to protect the muscle from excessive strain.

The follow-up part of the hypothesis (commands are sent to fusimotor neurons) and the length-servo part (muscle length is the regulated variable of the motor servo) were combined as follows. The intrafusal shortening produced by the  $\gamma$ -command was supposed to establish a reference length, creating an error signal at the summing node, and the servo was then supposed to reduce this error toward zero by producing a controlled amount of extrafusal shortening. A high gain in the length-feedback pathway would make the amount of extrafusal shortening closely approximate the prior shortening of the intrafusal fibers and also be relatively independent of disturbances produced by changes in mechanical load. This supposed reduction of the dependence of movement on the loading conditions is generally referred to as *load compensation*. Both the follow-up part and the length-servo (or load compensation) part of Merton's hypothesis have been challenged by subsequent experimental data and by alternative hypotheses.

#### Spindle Receptors as Model-Reference Error Detectors

Experimental studies stimulated by Merton's hypothesis suggested that command signals for movement are sent to  $\alpha$ - and  $\gamma$ -motoneurons more or less simultaneously (the evidence is reviewed briefly in  *$\alpha$ - $\gamma$ -Relations*, p. 308, and in more detail in the chapter by Matthews in this *Handbook*). This situation, termed  *$\alpha$ - $\gamma$ -linkage* by Granit (96) and  *$\alpha$ - $\gamma$ -coactivation* by Phillips (236), is captured by Figure 8 if it is assumed that  $\alpha$ - and  $\gamma$ -commands arrive as a combined pair, either in parallel pathways or in branches of the same input fibers.

Given this pattern of motor command, the dual responsiveness of spindle receptors to intrafusal contraction and muscle stretch provides a natural mechanism for a model-reference calculation (see *Control Configurations*, p. 262), if one also postulates that the intrafusal muscle represents a model of the controlled system, consisting of the main muscle and its load. In this view the forcing function to the controlled system ( $x_1$  in Fig. 7A and Fig. 8) is conveyed by action potentials in  $\alpha$ -fibers innervating the main muscle, whereas the forcing function to the model ( $x_2$ ) is conveyed by signals in  $\gamma$ -fibers that innervate intrafusal muscle. Movements are produced by sending signals in both alpha and gamma motor axons. If the subsequent shortening of the main muscle is equal to the shortening of the intrafusal muscle, the stretch applied to the sensory zone should not change nor should spindle discharge change. This represents the null condition described earlier in which actual output equals model output, and the reference error in Figure 7 is zero. In contrast, an unusually large load would interfere with

the shortening of the main muscle, causing a reference error in the form of increased discharge of spindle afferents. Conversely, an unusually small load would lead to an excess of extrafusal shortening, causing a reference error expressed as a decrease in spindle firing. It is clear from this discussion that the null condition of no change in spindle discharge should occur for some particular intermediate value of load, and loads differing from this should result in reference errors.

The movement-control hypotheses described in the following two sections are based on the initial assumption that muscle spindles function to detect reference errors in muscle length.

#### Conditional Feedback and Servo Assistance

The concept of  $\alpha$ - $\gamma$ -linkage led to a revision of the follow-up servo hypothesis that Matthews (195) referred to as *servo assistance*, for reasons to be explained below in this section. Essentially the same idea had been outlined briefly in the classic paper on the reafference principle published by Von Holst and Mittelstaedt in 1950 (285), and it also conforms with the suggestions offered in 1952 by Kuffler and Hunt (163). In the engineering literature this mode of control has been called conditional feedback, which, as discussed in *Control Configurations*, p. 262, and illustrated by option 1 in Figure 7A, is a subcategory within the general class of control systems termed model reference. Let us consider the rationale for these basically similar ideas.

The term conditional feedback stems from the fact that feedback in a system of this type vanishes when there is no reference error—thus a nonzero feedback signal is conditional upon the occurrence of an error. Similarly, the term servo assistance implies that the servo loop is quiescent when there is no error and becomes operative as part of the response to an error. For example, movement against an unusually large load would produce a reference error in Figure 8, and the latter should excite motor neurons to produce an enhanced force in compensation for the added load. In contrast, movement against a normal load would result in equivalent intrafusal and extrafusal shortening, no reference error, and no servo assistance.

The condition of a vanishing reference error requires some explanation. What is really meant is that spindle receptor discharge remains constant at some finite level, rather than actually dropping to zero impulse/s. This biasing of discharge about some elevated level is important, since it permits the detection of bidirectional reference errors that result from unusually small versus unusually large loads.

One of the interesting consequences of the conditional-feedback configuration is that the effects of disconnecting the feedback signals, as for example in the dorsal rhizotomy procedure, should be much less devastating than they would be in a more conventional

feedback system. Assuming there is some simple compensation for the withdrawal of excitatory bias provided by tonic spindle discharge, the rhizotomized subject should be able to make movements against usual loads without any modification in the  $\alpha$ -commands (Fig. 8) sent from higher centers. Problems would result mainly when unusually large or small loads are encountered. Thus, the notion of conditional feedback may help to explain the considerable motor abilities that remain after dorsal rhizotomy.

#### *$\beta$ -System and the Possibility for Zero Sensitivity*

The skeletofusimotor, or  $\beta$ -configuration, shown in Figure 9 also has the potential to operate as a model-reference system. The reader will recall that  $\beta$ -motoneurons branch to innervate both extrafusal and intrafusal muscle fibers. Until recently this type of innervation was thought to be prominent only in amphibians and reptiles, but more recent work discussed in **IMPLEMENTATION OF MOVEMENT COMMANDS**, p. 308, indicates that  $\beta$ -innervation probably occurs regularly in mammals as well, along with  $\alpha$ - and  $\gamma$ -innervation.

In order for the  $\beta$ -configuration to provide conditional feedback one must again postulate that the intrafusal muscle functions as a model of the controlled system, the extrafusal muscle and its load. In this case the main forcing function is conveyed by the branches of  $\beta$ -motoneurons innervating extrafusal muscle fibers, whereas the forcing function to the model is conveyed by the branches innervating intrafusal muscle (compare Figs. 7A and 9). As with the  $\alpha$ - $\gamma$ -configuration shown in Figure 8, the reference error is assumed to be the difference between the amounts of intrafusal and extrafusal shortening. Feedback is "conditional," since under some ideal (presumably the usual) loading condition the resultant reference error registered during movement should be zero.

The  $\beta$ -configuration has another interesting feature

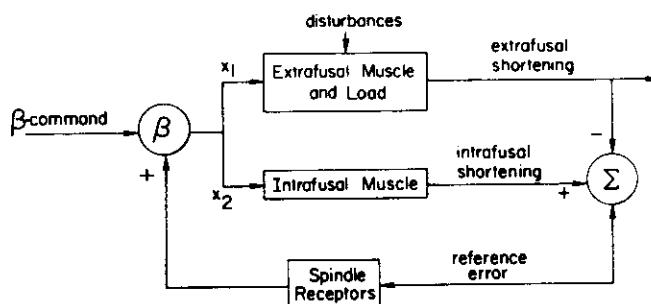


FIG. 9. Block diagram illustrating the organization of  $\beta$ -innervation. Forcing functions from  $\beta$ -motoneurons promote both extrafusal and intrafusal shortening. As in Figures 7 and 8, spindle receptors are shown detecting the difference between intrafusal and extrafusal shortening, a reference error. The positive-feedback loop from  $\beta$ -motoneurons through intrafusal muscle and spindle receptors back on  $\beta$ -motoneurons raises the possibility of zero-sensitivity operation.

built into its basic structure, a positive-feedback loop from  $\beta$ -motoneurons to intrafusal muscle through spindle receptors back to  $\beta$ -motoneurons. A theoretical analysis of the system (127) has demonstrated that with this positive-feedback pathway, zero-sensitivity operation (option 1 combined with option 2 in Fig. 7A) could be achieved, provided the gain of this loop is unity. At first sight this seems to be an advantage that the  $\beta$ -configuration has over separate  $\alpha$ - and  $\gamma$ -innervation. It has also been shown (127), however, that zero-sensitivity operation could occur if spindles projected back on  $\gamma$ -motoneurons. Autogenetic  $\gamma$ -projections were once rejected but are now being reconsidered (cf. ref. 62, 227). Spindle afferents also project to the brain where reference errors might be used to compute adaptive modifications (option 3 in Fig. 7A, along the lines discussed in **CONTROL THEORY CONCEPTS**, p. 258).

#### *Stiffness Regulation* la longueur du muscle le servira de référence du moteur

A major assumption in each of the hypotheses discussed up to this point is that muscle length is the regulated variable of the motor servo. This assumption is not well supported by current experimental evidence, however, as reviewed later, nor does it fit well with the presence of negative force feedback from tendon organs (127). These considerations together with supporting data led Nichols and Houk (226) to propose instead that stiffness, which is a ratio of force change to length change, may be the regulated property of the motor servo. A simplified graphic analysis of a hypothetical stretch reflex will serve to introduce the concept of reflex stiffness in relation to its neural and mechanical origins.

Figure 10 illustrates the three major components of static response that occur when an added load lengthens a muscle to elicit a stretch reflex. The first, a muscular component, is purely mechanical and is associated with the positive slope of the muscle length-tension curve (shown dashed in Fig. 10). The second, a length-feedback component, is mediated by the spindle pathway and consists of a facilitation of motor output that tends to increase force. The third, a force-feedback component, is mediated by the tendon organ pathway and consists of an inhibition of motor output that tends to decrease force. The actual change in force is the result of these three components, as illustrated by point d in Figure 10. This overall motor servo response is characterized by the slope of the heavy line segment a-d, which is called *reflex stiffness* and is given by

$$\frac{\Delta f}{\Delta x} = \frac{F_1 - F_0}{L_1 - L_0} \quad (10)$$

where  $\Delta f$  represents the resultant force increment produced by a stretch  $\Delta x$ .

It is apparent from Figure 10 that length feedback acts to increase reflex stiffness, which improves the

Perturbation provoque une variation dans la longueur et donc la force musculaire et le ~~système~~ ~~balance~~ entre de deux et le ~~charge~~ de la réponse motrice correspond à la balance entre <sup>CHAPTER 8: CONTROL OF LENGTH AND TENSION</sup> ~~les deux~~ <sup>267</sup> et les ~~récepteur~~ ~~mechanismes~~ (~~comme l'ouverture~~)

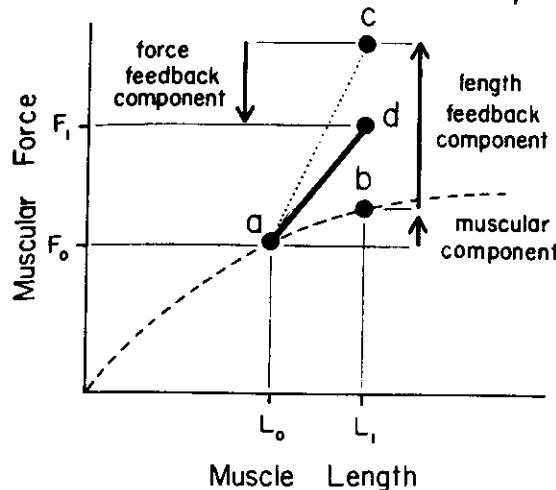


FIG. 10. Component analysis of hypothetical stretch reflex. Stretch from  $L_0$  to  $L_1$  causes force to increase from  $F_0$  to  $F_1$ . Reflex stiffness is defined by the slope of the line segment a-d equal to  $(F_1 - F_0)/(L_1 - L_0)$ . The diagram dissects this overall response into 3 components: the muscular-mechanical component arises from the length-tension properties of the muscle (dashed curve), the length-feedback component originates in spindle discharge and increases stiffness, and the force-feedback component originates in tendon organ discharge and decreases stiffness. [From Houk (127a). Feedback control of muscle: a synthesis of the peripheral mechanisms. In: *Medical Physiology* (13th ed.), edited by V. B. Mountcastle. St. Louis, MO: The C. V. Mosby Co., 1974.]

rigidity of length regulation in analogy with a stiff spring. A given load disturbance causes less disturbance in length if stiffness is high. In contrast, force feedback acts to decrease reflex stiffness, which is the same as increasing the compliance. This improves force regulation but interferes with length regulation, since a given load disturbance causes a greater disturbance in length if compliance is high. It is clear from this example that length and force cannot both be well regulated at the same time. In analogy with the discussion in *Regulated Variables and Properties*, p. 262, it would appear that combinations of length and force feedback might serve to regulate some property rather than each variable independently, and the theoretical analysis of this problem provided by Nichols and Houk (226; also see 128) suggests that the regulated property should be stiffness. A simplified version of this derivation will be given here.

Load perturbations ordinarily provoke changes in both muscle length and muscle force. Whether or not this causes a change in motor output should depend on the balance between length and force feedback as shown in Figure 1 and summarized by the following equation

$$\Delta e = g_s \Delta x - g_T \Delta f \quad (11)$$

where  $\Delta e$  represents the change in motor output, which is taken to be the error signal of the motor servo. For simplicity we have used the constant parameters  $g_s$  and  $g_T$  to represent the respective gains of spindle afferent and tendon organ pathways, and we

have assumed that control signals remain at constant values. It is particularly instructive to consider the null conditions under which there would be no need for a compensatory change in motor output, in which case  $\Delta e$  would remain equal to zero. Substituting  $\Delta e = 0$  and solving for  $\Delta f$

$$\Delta f_i = (g_s/g_T) \Delta x \quad (12)$$

where the subscript  $i$  designates that this is an ideal force change that results in no error signal. The special significance of  $g_s/g_T$  will become evident.

The actual force change is made up of mechanical and neurally mediated components

$$\Delta f = K \Delta x + A \Delta e \quad (13)$$

where  $K$  represents the mechanical stiffness, which in the example of Figure 10 is simply the slope of the length-tension curve, and  $A$  is an activation factor that converts the error signal into a neurally mediated component of force change referred to as reflex action (cf. *Dynamic Features of Force Development*, p. 297; and Fig. 35). To obtain Equation 12 we assumed an absence of any change in motor output, so we should also set  $\Delta e = 0$  in Equation 13

$$\Delta f_m = K \Delta x \quad (14)$$

where the subscript  $m$  designates that this is a purely mechanical response. The postulated perfect cancellation of length feedback by force feedback ( $\Delta e = 0$ ) will occur only if the mechanical force  $\Delta f_m$  happens to equal the ideal force  $\Delta f_i$ . Comparison of Equations 12 and 14 indicates that this condition is met if the mechanical stiffness of the muscle happens to be equal to the ratio  $g_s/g_T$ .

This ratio of gain factors has been called the *regulated stiffness*, since it represents a reference ratio that should determine the sign and magnitude of compensatory changes in motor output. Thus whenever the actual mechanical stiffness is less than this ratio, there is a deficit in force feedback,  $\Delta e$  in Equation 11 becomes positive, and the resultant input to the muscle tends to restore stiffness toward the reference ratio. Conversely, whenever mechanical stiffness is greater than this ratio, there is an excess of force feedback,  $\Delta e$  becomes negative, and the resultant decrease in motor output again tends to restore reflex stiffness toward the reference ratio. It is noteworthy that the regulation of this ratio property is predicted to result from a linear summation of excitatory and inhibitory signals at the level of the motoneuron. No fancy calculations are required.

The hypothesis for stiffness regulation does not eliminate the need for hypotheses concerning movement control. On the contrary, the two can be readily integrated (127). For example, the addition of a force-feedback loop to Figure 8 gives rise to a system in which stiffness is regulated while movements are controlled by  $\alpha$ - and  $\gamma$ -commands, in any combination. The major modification of the movement hypotheses

is that command signals now control the threshold of the stretch reflex rather than commanding a particular length. This, in fact, is quite consistent with the summary model of the motor servo described in the next section.

### Summary Model

The participation of the motor servo in complex motor acts is best understood with the aid of an overall model that concisely summarizes input-output properties. While summary models can be derived by combining models of the component parts, the more direct approach is to formulate them on the basis of input-output data. The account given here derives mainly from the work of Fel'dman (72, 73), stimulated by the earlier experiments of Matthews (193, 194) and supported by numerous studies that are reviewed later in this chapter.

Although the mechanical load is an integral part of the motor servo (Fig. 1), system characterization is simplified by considering the neuromuscular components (enclosed by dashed lines in Fig. 1) and the mechanical load separately. Since the properties of the load are easily modeled by well-known equations, the main problem becomes one of discovering a mathematical expression of the form

$$f = F[x, c_i] \quad (15)$$

that details the input-output properties of the neuromuscular portion. Here muscle force  $f$  is chosen as the output whereon muscle length  $x$  and a composite control signal  $c_i$  serve as the major inputs. The dependence of motor servo properties on velocity and acceleration is not excluded since the operator  $F$  can perform differentiation of  $x$ .

The composite control signal  $c_i$  is actually a set of neural commands that act on the motor servo at several points and are potentially capable of an independent control of several parameters. However, Fel'dman (72) proposed, on the basis of input-output data to be reviewed later, that the central control of this system ordinarily is much simpler than this. Specifically he postulated that the potential dependence on several variables ( $x$  and the various  $c_i$ 's) in most cases reduces to a dependence on a single variable, the quantity  $(x - x_0)$  where  $x_0$  represents the threshold of the stretch reflex established as the net result of the combined actions of the individual  $c_i$ 's. Hence, Equation 15 can be rewritten as

$$f = \phi [x - x_0] \quad (16)$$

$\phi$  is an operator having the units of stiffness that specifies an "invariant" relationship between muscle force and length. While Fel'dman's work has emphasized the static properties of this force-length relation, a dependence on velocity can be included by permitting  $\phi$  to be a dynamic operator, representing the dynamic stiffness of the motor servo.

Since the actual dependence on velocity is rather

weak (see *Velocity Dependence and Damping*, p. 306), the salient features of the force-length relations can be portrayed by a set of curves on a plot of muscle force versus muscle length. The solid curve in Figure 11 shows the typical dependence of force on length observed experimentally under conditions in which it is safe to assume that control signals remained constant. The intersection with the abscissa, labeled threshold length, is the length the muscle assumes when it is unloaded, i.e., when  $f = 0$ . According to Fel'dman's hypothesis, neural control signals act mainly by altering this threshold,  $x_0$  in Equation 16, which has the effect of shifting the entire force-length relation along the abscissa, as shown by the dashed curve in Figure 11. The dotted curve is included to illustrate an alternative mode of control that is discussed in the next section.

The block diagram in Figure 12 indicates how Equation 16 can be combined with another equation

$$x = L[f_d - f] \quad (17)$$

where  $L$  represents load properties and  $f_d$  is a disturbance force, to evaluate overall motor servo performance. The specific form of  $L$  depends on the nature of the load. For example, if the load is inertial,  $L$  becomes

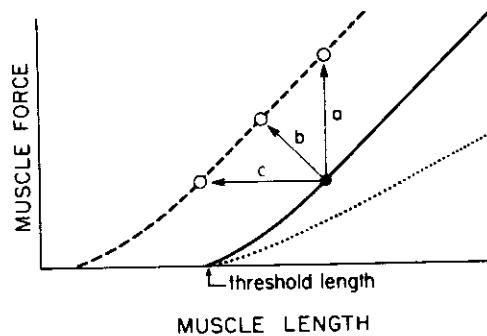


FIG. 11. Summary model of the motor servo. Solid line represents the static relationship between muscle force and length observed when control signals (cf. Fig. 1) remain constant. Control signals could act to change the threshold length (dashed curve) or to alter the slope of the force-length relation (dotted curve). The usual mode of control appears to be the former. Trajectories a, b, and c illustrate the load dependence of responses to a control signal. [From Houk (130). Reproduced, with permission, from Annu. Rev. Physiol., vol. 41. © 1979 by Annual Reviews, Inc.]

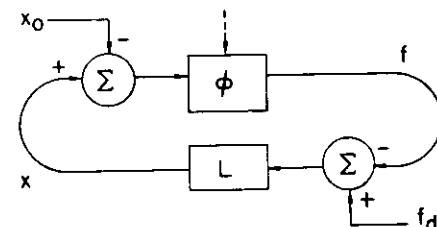


FIG. 12. Block diagram to illustrate the interaction between motor servo properties (represented by the operator  $\phi$ ) and the mechanical load. The operator  $L$  represents load properties,  $f_d$  is a disturbance force,  $f$  is muscle force,  $x$  is muscle length, and  $x_0$  is the threshold length of the strength reflex (Fig. 11) established by central motor commands.

division by a constant, the mass, and double integration; in the case of a spring load,  $L$  is a constant multiplier, the compliance of the spring.

The trajectories labeled a, b, and c in Figure 11 provide a graphic example to illustrate an important point—both muscle length and force are determined by the properties of both the load and the neuromuscular portion. The assumed input is a neural control signal that causes the illustrated shift in the force-length relation. The trajectories show the resultant changes in muscle force and length under three loading conditions: a) when pulling on an immovable object, b) when pulling on a spring, and c) when pulling against a constant disturbance force. Note that it would be inaccurate to say that the neural signal controls either length or force. Instead it controls the threshold length, and the particular force and length that result depend on the interaction between the properties of the motor servo ( $\phi$ ) and the load ( $L$  and  $f_d$ ).

#### *Adaptive Models*

It is well documented that a human subject can modify his responsiveness to mechanical disturbances based on prior instructions, such as "make your arm rigid" or "relax." The modifications are properly considered to be the product of an adaptive mechanism in the brain that controls the motor servo. The result is an alteration in the effective stiffness presented to mechanical loads that apparently meets changing requirements for reactions to the external environment (e.g., a compliant stiffness is advantageous for body suspension, whereas a rigid stiffness assists positional control). Two rather different models have been proposed to explain the neural mechanism for this type of adaptation.

Hammond (114) suggested that adaptation is achieved by altering the gain of transmission through length-servo pathways, a control strategy referred to as parametric adaptation. Coupling this idea with the hypothesis for stiffness regulation reviewed earlier, one can postulate that a controlled increase in the gain of length feedback ( $g_s$  in Eq. 12) would make the regulated stiffness more rigid, whereas an increase in force feedback ( $g_T$ ) would make it more compliant. These gain changes could be produced by presynaptic modulation or by gating inputs to interneurons in reflex pathways. Another interesting possibility is a selective control of the dynamic component of stiffness by activating dynamic fusimotor neurons ("adaptive control" in Fig. 8).

The hypothesis for a parametric adaptive control of the motor servo is contrary to (though not completely excluded by) the summary model discussed in the previous section, since it involves a change in the shape of the force-length relation (a hypothetical example of which is shown by the dotted curve in Fig. 11) rather than a simple shift of an invariant curve along the length axis (the dashed curve). However, the

possibility can be included in the summary model by postulating a second functional category of control signal, the unlabeled dashed input in Figure 11. This neural input is assumed to be an adaptive control that modifies  $\phi$ , the dynamic stiffness of the motor servo, via the mechanisms discussed in the previous paragraph. To avoid confusion, throughout the remainder of this chapter we use the term *motor command* to provide specific reference to the first and more usual type of control signal, the one that sets the threshold length in Figure 11.

An alternative hypothesis concerning the mechanism that adaptively controls the motor output is the two-stage model shown in Figure 13 (129, 130). Here the autogenetic reflex pathways of the motor servo are collectively represented by the box labeled Continuous Processor, whereas motor commands sent to the motor servo are postulated to be generated by a stimulus-response (S-R) processor. According to this model, adaptive responsiveness resides in the S-R processor rather than in the motor servo.

Unlike the operations of the motor servo, those of the S-R processor presumably do not employ continuous feedback; instead, it is assumed that afferent signals are analyzed primarily to detect the occurrences of environmental stimuli. Once detection is achieved, the process is triggered to release open-loop motor commands that control the motor servos regulating the activities of individual muscles. In other words, the S-R processor represents a central mechanism that generates motor commands that control reaction-time movements.

The adaptive controls applied to the S-R processor are presumed to be capable of altering either the particular sensory cues required to trigger a given response or the quantity or quality of the response released by any given sensory cue. One imagines a set of neural detectors that can be tuned to respond to different sensory cues or to internal commands; the initiatory signals from these detectors might then be used to trigger transitions in the state of a neural network that controls the levels of descending motor commands.

One of the interesting possibilities contained in this type of model is the potential for operating on the basis of the feedforward principle discussed earlier (cf. Fig. 4). Environmental stimuli associated with mechanical disturbances could be detected before they have had a chance to modify posture or movement and so used to trigger a compensatory motor command that counteracts a disturbance as, or even before, it develops.

#### *Summary*

Several of the models of the motor servo reviewed in this section focus on alternative roles for the fusimotor input to muscle spindles in movement control. While these hypotheses have generally included the assumption that autogenetic feedback functions to

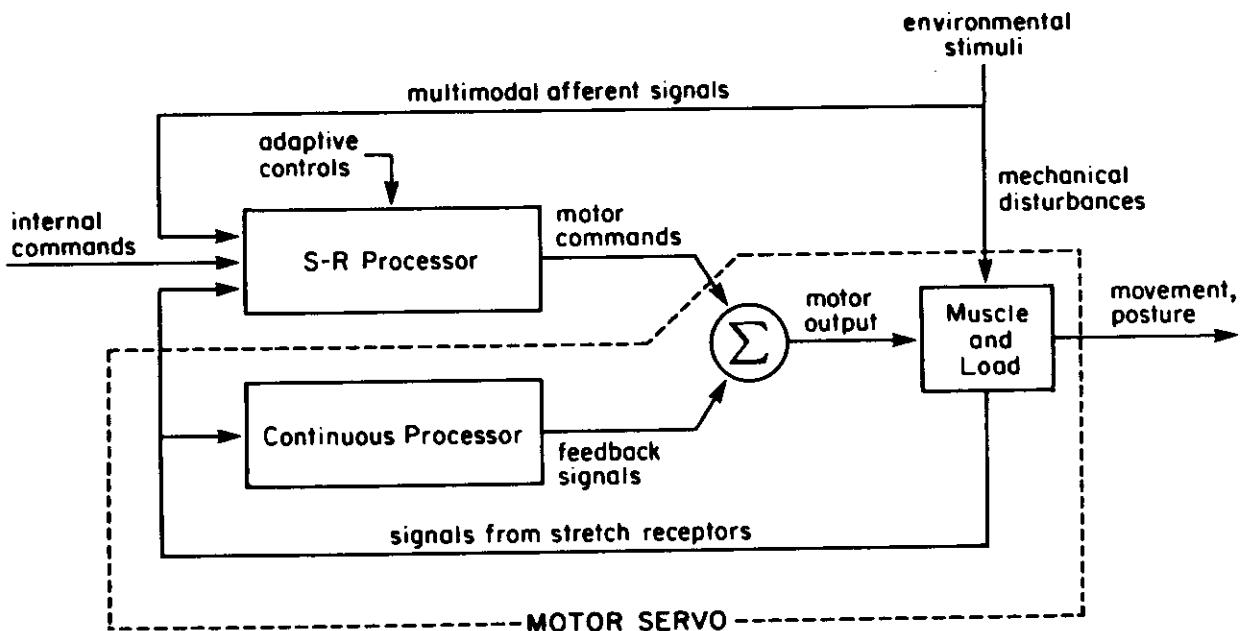


FIG. 13. Two-stage model of adaptive motor control. Motor output signals are assumed to be generated by 2 parallel processes that have rather different characteristic properties. Continuous Processor is assumed to operate in an analogue fashion, combining inputs from stretch receptors to produce continuous feedback compensation. The S-R (stimulus-response) Processor is assumed to operate more like a logical device, using sensory cues to trigger the release of preselected motor programs in a discontinuous fashion. Adaptive control is assumed to result mainly from the establishment of flexible S-R relations rather than from alterations in the gain of the continuous-feedback loops. [From Houk (129a). *Posture and Movement: Perspective for Integrating Sensory and Motor Research on the Mammalian Nervous System*, edited by R. E. Talbott and D. R. Humphrey. New York: Raven, © 1979.]

control muscle length, they can be modified to include other alternatives. One such alternative described here in some detail is the hypothesis that length and force feedback combine to regulate the stiffness of the motor servo. We also describe a summary model of the motor servo that is based on empirical input-output data, and we briefly discuss how the adaptive control of motor servo properties could be accomplished either by parametric gain control or by establishing versatile stimulus-response relations.

#### MUSCLE MECHANICAL STIFFNESS

An adequate assessment of the regulatory hypotheses discussed in the previous section requires knowledge of the manner in which a muscle responds to stretch and release in the absence of neuroregulation. There are deficiencies in muscle mechanical properties, and these deficiencies must be understood as a preface to an evaluation of the efficacy of the regulatory actions that compensate for them. The approach taken here is to describe the properties of skeletal muscle in terms of its mechanical stiffness and to review the dependence of stiffness on the initial conditions and on the parameters of length change. The reader is referred to the chapter by Partridge and Benton in this *Handbook* for a more general account of muscle physiology.

#### Stiffness Definitions

Stiffness is always represented by a force-to-length ratio, but since this ratio can be assessed in different ways, under either static or dynamic conditions, one needs to provide some alternative definitions. *Static stiffness* is the ratio of the increment (or decrement) in force divided by the corresponding increment (or decrement) in length measured under steady-state conditions. There are two useful measures of dynamic stiffness. *Incremental dynamic stiffness* ( $\Delta f/\Delta x$ ) is the ratio of force increment to length increment measured under transient conditions, whereas *instantaneous stiffness* ( $df/dx$ ) is a dynamic infinitesimal measure calculated from the slope of a force-length trajectory. Instantaneous stiffness ( $df/dx$ ) is equivalent to the slope of a time plot of force ( $df/dt$ ) during the constant velocity phase of ramp changes in length. In addition it is often convenient to use the term stiffness in a larger sense, with reference to an entire force-length relation. For example, the stiffness operator  $K$  in

$$f = K[x] \quad (18)$$

converts a length variable  $x$  into a force  $f$ .

If  $K$  in Equation 18 were a constant multiplier, the properties would be like those of an ideal (linear and undamped) spring. In this case all of the above men-

tioned measures of stiffness would yield the same result, the value of the spring constant  $K$ . Furthermore none of the measures of stiffness would depend on the initial length or force or on other initial conditions. However, muscle is both dynamic and highly nonlinear, and as a consequence its stiffness varies considerably depending on the velocity and amplitude of length change and on the initial conditions. Correspondingly, the reader should not expect this chapter to provide any unique values for stiffness. Instead the purpose of the chapter is to document the various conditions under which muscle stiffness varies.

### Length Dependence

Since length-tension curves are usually constructed from measurements of the steady force developed at each of several lengths, the slopes of these curves provide one way of assessing the static stiffness of the muscle. Figure 14 was prepared to provide a summary illustration of a typical set of length-tension curves for skeletal muscle. The active + passive curve represents a force-length relation for a maximally activated muscle—all motor units recruited and discharging at high rates. It is apparent from the slope of the curve that static stiffness is appreciable at intermediate lengths, but it decreases toward zero as the peak of the length-tension curve is approached, in spite of the fact that the force there is highest. In contrast the stiffness of the passive muscle is negligible at intermediate lengths

but becomes significant toward the end of the normal physiological range of muscle lengths.

The shaded region between the active + passive and the passive curves in Figure 14 is labeled the control zone, since it represents the range over which the CNS can modulate force by controlling motor output. Within this zone force is graded by the recruitment of motor units in the order small to large and by the modulation of the discharge rates of active units (see the chapter by Henneman and Mendell in this *Handbook*). Whenever force is varied by recruitment and rate modulation, there are simultaneous alterations in mechanical stiffness that occur in an obligatory fashion.

### Recruitment of Motor Units

The effect of recruitment on static mechanical stiffness has been studied by comparing the length-tension curves obtained when different numbers of motor units are electrically stimulated at physiological rates (138, 225). The results are in reasonable agreement with the simple hypothesis that recruitment adds contractile elements (muscle fibers) in parallel, although departures from this rule were noted for low levels of recruitment at short lengths.

The addition of contractile elements in parallel amounts to a scaling of the entire length-tension curve, in proportion to the level of recruitment. Thus if we let  $P[x]$  represent the active length-tension curve for

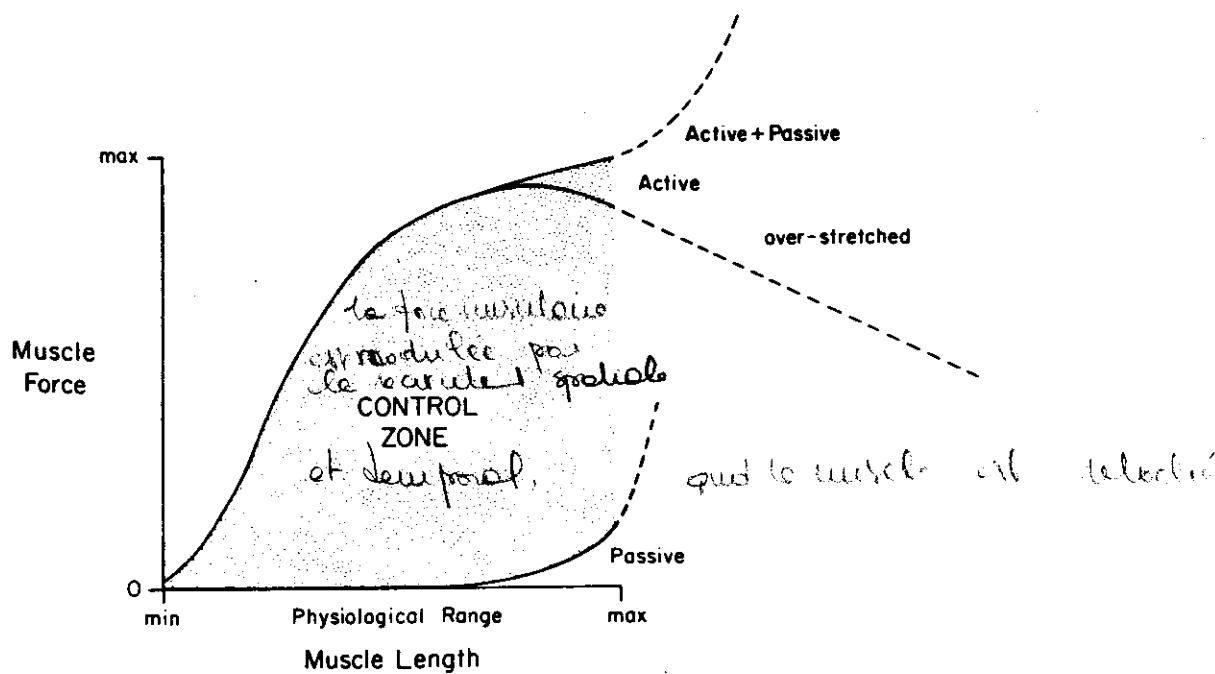


FIG. 14. Length-tension characteristic of skeletal muscle. *Active + Passive curve* shows the force produced by a typical muscle that is maximally activated, as a function of length throughout the physiological range. *Passive curve* shows the corresponding force when the muscle is entirely relaxed. *Shaded region* represents the control zone within which muscle force can be modulated by recruitment and rate modulation of motor units.

a fully recruited muscle, and if we use the ratio  $f_0/f_{\max}$  as a measure of the level of recruitment, the active component of muscle force can be written

$$f_a(x) = \frac{f_0}{f_{\max}} P[x] \quad (19)$$

where  $f_0$  is the initial force at any given initial length, and  $f_{\max}$  is the maximal force at that same length. In essence, the ratio  $f_0/f_{\max}$  serves as a measure of the cross-sectional area of myofibrils that have been recruited to activity.

The active mechanical component of static stiffness ( $K_a$ ) can be assessed from Equation 19 as follows

$$K_a(f_0, x_0) = \frac{\Delta f_a}{\Delta x} = \left( \frac{f_0}{f_{\max}} \right) \cdot \left( \frac{\Delta P[x]}{\Delta x} \right) \Big|_{x_0} \quad (20)$$

$K_a$ , like  $f_a$  in Equation 19, is directly proportional to the level of recruitment, and it also depends on the slope of the whole-muscle length-tension curve at the particular initial muscle length,  $x_0$ .

Total muscle force and total mechanical stiffness can be obtained by adding a passive component, whenever the latter is appreciable. Although the procedure of simply adding passive and active components to obtain total force is not precise, because the series elasticity of the tendon is commonly in series with both the passive parallel elasticity and the contractile mechanisms, errors that result from the simplification are seldom appreciable (cf. appendix in ref. 138, for an analysis that includes these corrections).

In conclusion, increases in force produced by the recruitment of motor units are closely analogous to adding prestretched springs in parallel. Consequently, recruitment produces an increase in mechanical stiffness that is approximately proportional to the increase in force.

#### Rate Modulation of Motor Units

The dependence of static stiffness on discharge rate has been studied by comparing length-tension curves obtained when a whole muscle is electrically stimulated at different rates within the physiological range (97, 103, 193, 225, 248). In several of these studies, distributed stimulation of ventral root filaments was used to mimic the asynchronous activity of motor units. Asynchrony normally insures smooth contractions even though discharge rates are well below the fusion frequencies of the individual units. The dashed curves in Figure 15 show the larger forces and the characteristic shifts of the length-tension curves to the left produced by higher rates of stimulation. The unbroken traces are dynamic trajectories that will be discussed in the next section.

Rack and Westbury (248) correlated their findings on whole muscle with the sliding filament model of contraction, based on histological measurements of sarcomere lengths. The curves obtained with stimulus rates of 50 impulses/s or higher correspond reasonably

well with the clamped-sarcomere length-tension curves obtained by Gordon, Huxley, and Julian (89), provided the somewhat longer length of thin filaments in mammals, as opposed to amphibians, is taken into account (40). However, at lower and more physiological stimulus rates the force at any given length was considerably smaller. This finding was attributed to bond turnover caused by internal shortening and lengthening movements within individual sarcomeres, since the latter were activated at rates below their fusion frequencies. Regardless of the specific mechanism, it is quite clear that low activation rates yield length-tension peaks at points beyond the region of maximum cross-bridge overlap, and the steep ascending portion of the curve can occur quite independently of the degree of filament overlap (225, 248).

Inspection of the slopes of the length-tension curves in Figure 15 illustrates that increasing rate can either increase or decrease muscular stiffness, or it can leave stiffness unaltered, depending on the particular rates and on the initial length about which the slope is assessed. For example, at the length corresponding to a 105° ankle angle, stiffness is low at 5 impulses/s, increases to a substantial value at 10 impulses/s, and then decreases once more at 35 impulses/s. While the corresponding variations are somewhat less if one holds constant the initial force, rather than the initial length, clearly there is no simple relation between stiffness and motor unit discharge rate.

In spite of these complexities, a linear model fit to the family of curves may provide adequate accuracy under appropriately restricted conditions. Thus the effects of discharge rate are sometimes approximated by a shift in the slack length of a linear spring

$$f = K(x - x_1) \quad (21)$$

where  $f$  and  $x$  represent force and length, the constant  $K$  is the stiffness of the spring and  $x_1$  is its slack length. Increasing discharge rate is treated as a reduction in  $x_1$ . As will be pointed out later, this model appears to be more accurate as a descriptor of the responses to slow, constant velocity stretches as employed by Grillner (103), than it is for static length-tension properties such as those illustrated in Figure 15.

#### Instantaneous Stiffness and Short-Range Elasticity

Instantaneous stiffness can be assessed from the slopes of the solid trajectories in Figure 15, which are plots of instantaneous force as a function of length during periods of stretch at constant velocity (152). These stretch responses followed an isometric period during which force built up to various initial values (the  $x$ 's in Figure 15), dependent on initial muscle length and stimulus rate as discussed earlier. The slopes of the trajectories that take off from each initial point represent the instantaneous stiffness that prevails after different amounts of stretch. It is apparent

### *Instantaneous Stiffness Beyond the Short-Range Region*

The transition from the short-range elastic region is marked not only by a reduction in stiffness but also by a qualitative change. Prior to the transition, muscle mechanical stiffness is approximately constant at a given initial force and it is predominantly elastic. In other words, force change is proportional to and in phase with the length change and essentially independent of velocity. In contrast, stiffness develops a complex dependence on length, velocity, and stimulus rate after the transition, as illustrated by the trajectories in Figure 15 subsequent to the point at which yielding occurs. There appears to be a transient phase, which lasts longer at lower stimulus rates, followed by a convergence of the trajectories on what can be recognized as a dynamic length-tension curve. The latter lies entirely above the static length-tension curve at high stimulus rates, as would be expected if a frictional force added to that static force. At lower stimulus rates, however, the dynamic curve falls below the static curve at short lengths, which on a phenomenological level represents negative friction, and rises above the static curve at longer lengths. These particular alterations tend to make the dynamic length-tension curve at stimulus rates in the vicinity of 10 impulses/s more linear than the static one, which may be why the curves reported by Grillner (103) appear more linear than those reported by other authors.

All of the trajectories in Figure 15 were obtained during stretch at the same velocity, one within the range encountered by this muscle during walking. Higher stretch velocities give rise to larger forces, although the differences are small and the dependence on lengthening velocity clearly falls short of being proportional (151, 152, 225). Shortening always results in a fall in force from the isometric value at any given length, even at low stimulus rates, and the change seen is typically larger than that associated with an equivalent velocity of lengthening. The results during shortening are approximately fitted by the well-known force-velocity equation of Hill (121), which has been repeatedly shown to be quite accurate for shortening during higher rates of stimulation.

#### *Ramp Responses: Transient Properties and Nonlinearity*

Transient responses to ramp-and-hold changes in length provide an effective means for studying both dynamic and static properties and the transition between the two that occurs during the plateau phase of the ramp. Figure 16 shows examples of transient responses to stretch, superimposed upon isometric contractions for three different rates of stimulation. Muscle linearity can also be assessed if ramp amplitude and direction are varied while holding the duration of the constant velocity phase fixed. Figure 17, which includes only the incremental portions of force records

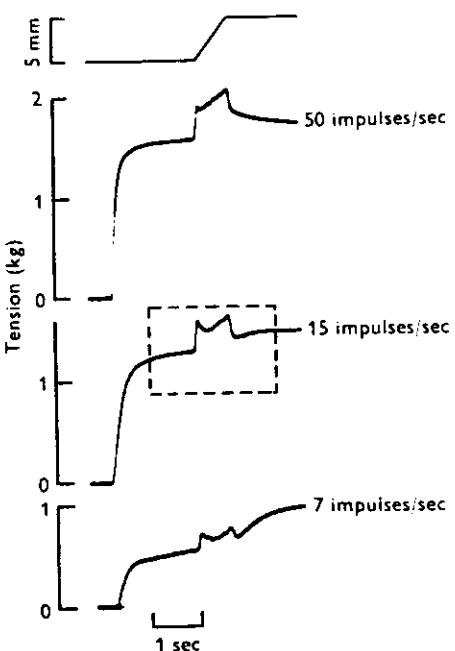


FIG. 16. Responses of cat soleus muscle to ramp-and-hold stretch (5 mm at 7.2 mm/s) applied while the muscle was being stimulated at each of 3 rates. Dashed box encloses the incremental transient response, shown as a function of ramp amplitude and direct. Figure 17. [Adapted from Joyce et al. (152).]

(the part enclosed within the dashed box in Fig. 17) illustrates the manner in which the transient response depends on the amplitude of stretch (records on left) and release (records on right) when the stimulus is 13.3 impulses/s. The release responses are easier to understand and therefore are considered first.

Responses to release are dominated initially by high dynamic stiffness of the cross bridges and by the much lower static stiffness associated with the slope of the length-tension curve. The dynamic static transition is a phase of tension redevelopment initiated by the breakdown of cross bridges and motored by their reformation at somewhat shorter comere lengths. It is apparent from Figure 17 that the time course of this transitional process is faster for larger amplitudes of release, which is a nonlinear feature. So although the response prior to ramp plateau (first vertical marker in Fig. 17) scales in direct proportion to the magnitude of the ramp, the criteric linearity, the response evaluated 1 s after ramp (second vertical marker) does not. This is a slight nonlinear feature. The 1-s response is more sensitive to amplitude for small ramps than it is for large ramps. Overall, responses to release demonstrate appreciable dynamic properties but only modest departures from linear behavior.

The responses to stretch are also dominated initially by the high stiffness of cross bridges and ultimately by the low stiffness associated with the length-tension curve. However, the transition between the two is complicated by the yield in force that occurs during

stretch and by the dip in force that occurs just after ramp plateau. It is apparent from Figure 17 that the yield occurs earlier when the size (and also the velocity) of the ramp are increased. This is consistent with the notion that the range of high stiffness prior to cross-bridge turnover corresponds with a certain amplitude of stretch. The actual change in latency is somewhat less than predicted on this basis, which documents the secondary dependence of this range on velocity.

After yielding there must be a slippage between thick and thin filaments that continues for the remainder of the constant velocity phase of the ramp. Remarkably the force increment toward the end of this phase varies by less than a factor of 1.5 in the face of a 17-fold increase in the size of the ramp. Although performance up to the time of yield is quite linear, after the yield it is highly nonlinear as evidenced by the failure to scale in proportion to the amplitude of the input. In the period just after ramp plateau, slippage between thick and thin filaments apparently becomes particularly marked, giving rise to the dip in force that sets this variable to a value below the ultimate isometric force at the new length. Phenomenologically the dip corresponds to a phase of negative incremental stiffness and a continuation of highly nonlinear behavior.

The overall effect of stimulus rate on the transient response is best understood in terms of three component effects. The first is that higher rates promote more cross-bridge formation, which tends to make response scale in proportion to the initial force (Fig. 16). Proportional scaling is generally valid only for the

region of short-range elasticity, but in special cases it may hold approximately for later phases of response (122, 226). A second effect concerns the changes in the shape of the length-tension curve produced by increasing rate (Fig. 15), which can either increase or decrease static stiffness as discussed previously. The third effect concerns the manner in which rate changes affect the transition from short-range elasticity to the static stiffness of the length-tension curve. As illustrated in Figure 16, lower rates result in a disproportionate fall in the force developed during the constant velocity phase after yielding and in the immediate postramp period, thus accentuating the yield and dip in responses to stretch and slowing the redevelopment of isometric force in responses to release. The latter effect also appears as a slowing of the initial development of isometric tension at lower stimulus rates in Figure 16.

The less drastic yield and the absence of a dip in the 50 impulses/s record in Figure 16 probably results from an enhancement of the rate of cross-bridge formation promoted by a higher sarcoplasmic calcium concentration. Though this degree of enhancement is atypical for the soleus muscle when activated at its normal tonic rates, it may actually be more typical for other muscles such as the cat gastrocnemius (249). High levels of activation reduce or eliminate the phases of negative stiffness noted earlier, but this does not alter the fact that the stretch response during the transitional period is highly nonlinear.

In contrast to these complex alterations in transient responses produced by rate modulation, the effect of recruitment is relatively simple. As with the static case

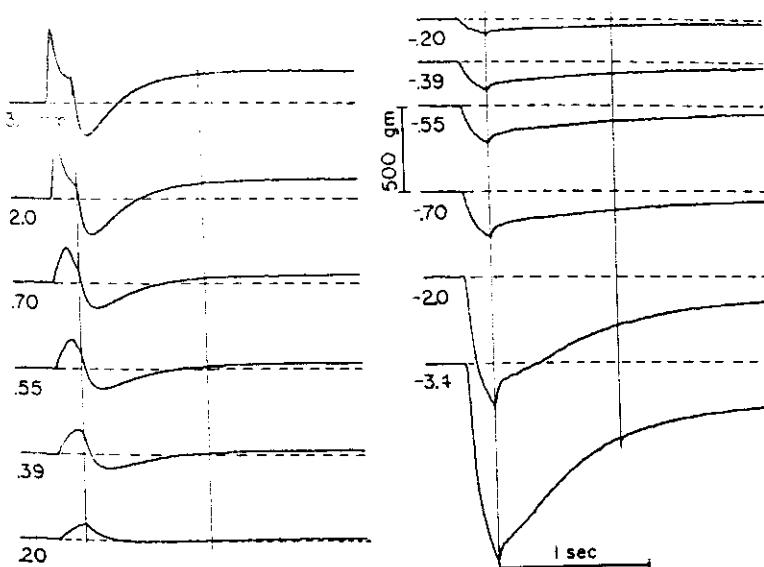


FIG. 17. Incremental transient responses of cat soleus muscle obtained with different amplitudes of stretch and release. Muscle was stimulated at 13.3 impulses/s before and throughout each force record. First vertical marker indicates the time of ramp plateau (160 ms) and the second indicates 1 s after ramp initiation. Note that the changes in force are not scaled versions of each other as they would be for a linear system. [From Nichols (225).]

discussed earlier, recruitment effects on transient responses are quite well accounted for by the assumption that the muscle fibers are functionally in parallel with each other, at least in the case of the soleus muscle where this has been tested (226). Thus the entire nonlinear transient response scales in proportion to the level of initial force.

#### *Natural Combinations of Recruitment and Rate Modulation*

Recruitment and rate modulation are ordinarily used in combination to grade muscle force. The available evidence suggests that recruitment is the more important mechanism at low levels of force, whereas rate modulation is more important at high force levels (214, 217). So one would expect appreciable variations in active muscle stiffness as force varies in the low range and perhaps less appreciable variations in the high force range. Nichols and Houk (226) evaluated the combined effects in the soleus muscle of the decerebrate cat by grading initial force with a crossed-extensor reflex after autogenetic reflexes had been abolished by transection of the ipsilateral dorsal roots. While there was a fair amount of scatter in the data, both dynamic and static phases of response varied approximately in proportion to the initial force throughout the lower half of the physiological range; it was not possible to study the upper half of the range of force gradation. This result suggests that Equations 19 and 20, though strictly applicable only to recruitment, may be sufficiently accurate for a first-order characterization of the variations in muscle force and mechanical stiffness that result from recruitment and rate modulation in combination, at least in the 0%-50% range of force gradation.

#### *Summary*

The inherent mechanical stiffness of skeletal muscle depends in a complex manner on the initial conditions and on the parameters of length change. Static stiffness is proportional to initial force when the latter variable is graded by recruitment. When force is graded by changing initial length or discharge rate, however, the dependence is more complex and is best summarized by the slopes of a family of length-tension curves. Within the short-range elastic region, dynamic stiffness (both incremental and instantaneous) is high in comparison with static stiffness, relatively independent of the direction and velocity of length change, and proportional to the initial force, whether the latter is graded by recruitment, rate modulation, or changes in initial length. This short-range elasticity yields, and stiffness decreases abruptly when the muscle is stretched at moderate to high velocities by more than about 2% of the physiological range of length change. The magnitude and time course of the decrease in stiffness depend in a complex manner on stimulus rate,

initial length, and the velocity and amplitude of stretch; the dependence on the level of recruitment is simply proportional to initial force. Alterations in stiffness also occur in association with release beyond about 2%, but the changes are less marked than those associated with stretch.

These variations in mechanical stiffness appear to represent deficiencies in muscle performance, which are partially compensated by motor servo actions, as described in later sections.

#### CENTRAL PATHWAYS

The output of the motor servo depends upon sensory input from spindle receptors and tendon organs to derive the appropriate error signal and to issue the appropriate corrective response (see *Stiffness Regulation*, p. 266). A necessary step in these derivations is the passage of this sensory information across one or more intervening synapses in the spinal cord. The purpose of this section is to review briefly the pathways by which muscle afferents influence spinal motoneurons, concentrating largely upon autogenetic connections; these connections are summarized in Figure 18. A detailed description of the general electrophysiology and connection of spinal neurons has been presented in the first volume in this *Handbook* section on the nervous system (35) and in the chapter by Baldissara, Hultborn, and Illert in this volume. Reciprocal Ia inhibitory effects and the segmental actions of Renshaw cells are considered in the same chapter.

#### *Primary Ending Projections*

The primary spindle (Ia) afferent projection to homonymous motoneurons has been studied extensively, [see reviews of Redman, (250); Burke and Rudomin, (35)]. In relation to the problem of motor servo action, the Ia-afferent volley that originates with the onset of rapid muscle stretch appears to be important in initiating an early onset of motor output. It is also likely that this initial, predominantly monosynaptic Ia input causes motoneurons to be recruited in a sequence consistent with the size principle (see the chapter by Henneman and Mendell in this *Handbook*). The basis of continuing motor output remains uncertain when it is observed, for example, during the maintained phase of a stretch-reflex response. There is little doubt that the monosynaptic Ia-projection is an important source of excitation; nevertheless, there are suggestions that other sources of excitation probably exist.

Although the possibility of polysynaptic Ia-projections has been advanced intermittently, the evidence has remained largely indirect in nature (6, 101, 140, 142, 153, 228, 241, 275). The relevant arguments regarding Ia polysynaptic input are based on the results of intracellular recordings in motoneurons and on the time course of motoneuronal discharge and force change in whole muscle.

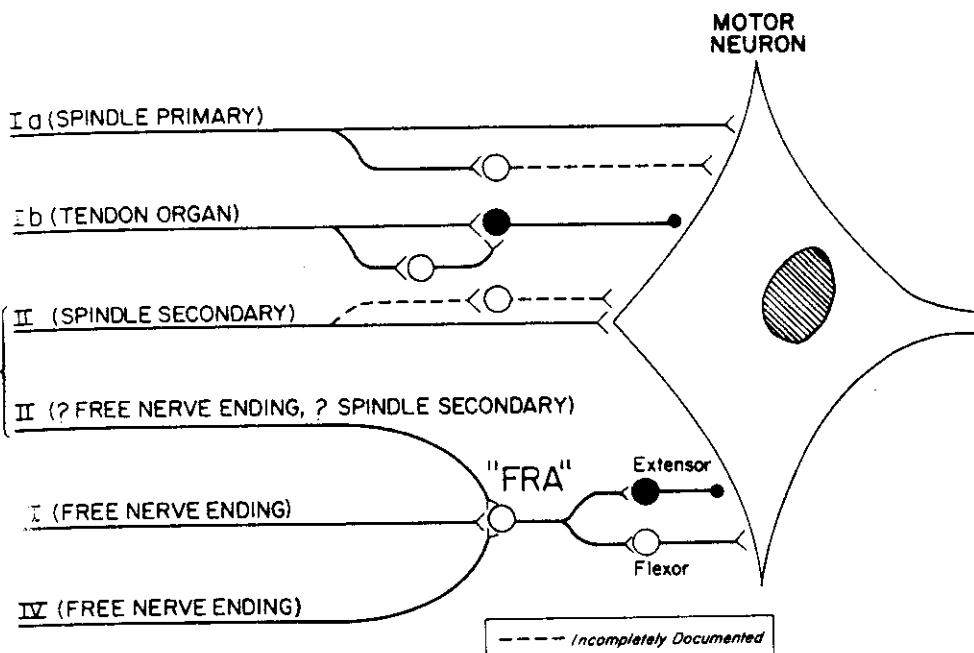


FIG. 18. Summary of the major autogenetic connections of muscle afferents to spinal motoneurons. Well-documented pathways such as Ia, Ib, and flexor-reflex afferents ("FRA") are shown as continuous lines. More speculative or incompletely documented connections are shown as dashed lines. ○, Excitatory interneurons; ●, inhibitory interneurons. Renshaw cells are omitted for simplicity.

illustrate the findings derived from intracellular recordings, Ia excitatory postsynaptic potentials (Ia EPSPs) arising at polysynaptic latency have been reported in a cat model of spasticity (228) and during fictive locomotion elicited in the spinal cat (261). The existence of an excitatory interneuron is also supported by studies in which combined muscle stretch and stimulation of the pinna produced a prolonged period of increased excitability of the extensor motoneurons that far outlasted the duration of either excitatory stimulus. This augmented excitability was attributed to posttetanic potentiation at an interneuron receiving convergent input from Ia-afferent and other sensory fibers (101). Repetitive electrical stimulation of Ia-afferents can also give rise to prolonged electromyographic activity that far outlasts the stimulus duration (140–142), implying that reverberative activity of Ia-interneurons might exist. Alvord and Fuortes (6) and later Tsukahara and Ohye (275) showed that during stimulation of the Ia-afferent input, motoneuronal firing probability was increased at latencies that were consistent with a polysynaptic Ia-projection.

Perhaps the most convincing evidence in support of polysynaptic Ia-projections is that derived from studies using longitudinal tendon vibration. The slow rise time and decay time of the force output during tonic vibration (110, 196), the lack of dependence of force output upon the detailed temporal properties of afferent input (200), and the profound depression of the tonic vibration reflex (TVR) by minute doses of barbiturate that are insufficient to impair monosynaptic transmission (153) suggest that polysynaptic Ia-pro-

jections may be important. While these findings would seem to implicate a Ia-interneuron, no Ia-interneuron has yet been identified that provides direct excitatory projections to the homonymous motoneuron pool (hence the incompletely documented designation in Fig. 18). A group of interneurons has been described that receives Ia-afferent excitation, and that appears to lie more laterally in the intermediate spinal nucleus than the well-studied inhibitory Ia-interneuron pool (see ref. 149). These neurons would seem to be excellent candidates, but their efferent connections have not yet been studied.

In spite of the lack of direct verification, a potentially significant contribution from excitatory Ia-interneurons to motor servo action remains likely.

#### Tendon Organ Projections

The existence of inhibitory actions of Ib-fibers on motoneurons has been recognized for some time, largely on the basis of intracellular recordings that showed inhibitory postsynaptic potentials (IPSPs) arising at latencies consistent with the existence of one or more intervening interneurons (56; see Fig. 18). Activation of the Ib-projection to extensor motoneurons has been consistently recorded as inducing disynaptic or trisynaptic inhibition, but the effects on flexor motoneurons are less clearly defined. For example, Eccles et al. (56) found rather few examples of Ib synaptic effects on flexor motoneurons. This deficit appears to have been a consequence of a lack of appropriate descending bias, because quite strong in-

hibitory potentials are revealed when Ib-afferent stimuli are conditioned with tetanic stimulation of the red nucleus (126). The experiments cited also revealed that many actions of Ib-fibers are not just reciprocally organized. Coupled excitatory and inhibitory effects, extending to muscles acting at quite distant joints, have been reported (56, 126).

A number of studies have reported the existence of neurons in the intermediate nucleus of the spinal cord that are sensitive to electrical activation of muscle nerves at Ib strength—these neurons could correspond with those producing the electrically induced IPSPs (57, 125, 288). Relatively few attempts to characterize the putative Ib-interneuron have been made using adequate stimulation of tendon organs as a means for activating the Ib-afferent input. Granit et al. (100) showed that the silent period observed during muscle twitch is mediated partly by tendon organ-induced postsynaptic inhibition. The study by Lucas and Willis (175) verified that a small-amplitude tendon tap was a selective stimulus to Ia-afferents, (179, 268) and then showed that larger tap amplitudes activated both Ia- and Ib-afferent fibers. Using the sensitivity to large-amplitude taps as a criterion, a substantial number of interneurons in the intermediate nucleus of the cat lumbosacral spinal cord were reported to receive Ib-afferent input (175). However, given the nonselective nature of the input, some residual doubt regarding this identification must persist. To this date no method for selective activation of Ib-afferents has been used, although there is promise in techniques based upon the procedure described by Jack and Roberts (146), in which the threshold of Ia-afferent fibers was increased by prolonged tendon vibration, allowing subsequent relatively uncontaminated electrical stimulation of Ib-fibers. The efferent projection of interneurons receiving Ib input has not been investigated, and its correspondence (or lack of correspondence) with the properties anticipated on the basis of intracellular recordings remains to be established.

It has recently been reported that there is an inhibitory autogenetic Ia-projection (77) that may well arise from interneurons receiving convergent Ia and Ib input. The functional significance of such mixed convergence patterns has not yet been explored.

#### *Projections From Secondary Endings and Group II Free Nerve Endings*

The traditional view of the central actions of group II afferents was based on classic monosynaptic testing techniques (172, 173) and on intracellular recordings obtained during electrical stimulation of muscle nerves (27, 60). The consensus has been that group II fibers originate almost entirely in secondary endings, and that secondary spindle afferent effects are mediated through interneuron pools that are inhibitory to extensor motoneurons and excitatory to flexors, features shared with many other high-threshold muscle and

cutaneous afferents. These apparently uniform central effects have led investigators to assign the group II afferent pathway to the broad class of flexor-reflex afferents [the "FRA" of Holmqvist and Lundberg, (124)] that is characterized by diffuse flexor activation and inhibition of extensors. Recently, this view has been challenged by a number of studies indicating that the central projections of group II afferents may not be uniform in their action, but may mediate two opposing types of effects.

The claim of dual excitatory and inhibitory actions of group II fibers is based largely on the results of innovative approaches to the central actions of muscle receptors. For example, several studies have utilized identified single spindle secondary afferent fibers, isolated in continuity from dorsal root fibers, in the manner of Mendell and Henneman (207, 208). The spike discharge on the single fiber is then used to synchronize an ensemble average of intracellular events—a spike-triggered average. Both Kirkwood and Sears (158, 159) and Stauffer et al. (266) have shown that distinct monosynaptic EPSPs can be extracted from background synaptic noise with this technique. The EPSPs are usually somewhat smaller than those generated by single Ia-afferent fibers (approximately 40–70 µV for primary afferents versus 8–30 µV for secondaries) but are of sufficient magnitude to provide a significant excitatory input. This monosynaptic projection is depicted as part of the group II secondary spindle input in Figure 18. Further evidence in support of excitatory group II projections has been derived recently using averages of synaptically mediated population potentials in the motor nuclei (180, 181). These potentials were measured using the proximal ends of sectioned ventral roots, and the averages were synchronized by the discharge of intact secondary afferent fibers. The latency and time course of the averaged potentials were not unlike those of monosynaptic EPSPs.

An earlier series of studies (197, 204) based largely on the use of vibration as a means of clamping the Ia-afferent input was the first to suggest that secondary spindle afferent fibers may contribute autogenetic excitation to extensor motoneurons. These studies depend on the finding that small-amplitude, longitudinal vibration of a muscle tendon acts as a selective stimulus to the primary ending, producing one-to-one driving of the ending for each cycle of vibration (24, 28). Under some conditions high-frequency tendon vibration induces a sustained muscle contraction, the TVR (196). Using this capacity of vibration to clamp Ia-afferent discharge, Matthews (197) reported that the vibration reflex and stretch reflex did not interact in a way that would have been predicted were they each to depend entirely on the Ia-afferent input. Specifically the increment in force evoked by vibration did not decline as the muscle length (and the resting discharge of the Ia-afferent) was increased. On the basis of these and other results, Matthews suggested

that the stretch-induced central excitation was not mediated entirely by the Ia-afferent pathway. The secondary spindle pathway was advanced as a possible source of this excitation, entirely on indirect grounds.

Further evidence supporting the existence of a second source of afferent excitation for motoneurons was provided by Westbury (287), McGrath and Matthews (204), and Kanda and Rymer (154) on the basis of experiments in which vibration was used to control the Ia-afferent input. Although these various experiments provided suggestive results, they did not directly identify the source of the excitation. Furthermore Jack and Roberts (147) have reported that one-to-one driving of Ia-afferent fibers by each vibration cycle is disrupted when the muscle is contracting and then stretched over several millimeters. This disruption is especially prominent during states of varying dynamic fusimotor bias.

The hypothesis that group II afferents excite extensor motoneurons is further complicated by the long-standing observation of inhibition of extensor motoneurons when group II fibers in muscle nerves are stimulated electrically. Two broad hypotheses have been advanced to reconcile the various results.

The first hypothesis (197, 199, 259) suggests that two separate sets of group II fibers with separate central connections exist. Many muscle nerves are known to contain substantial numbers of group II fibers that might not originate in secondary spindle receptors (see, e.g., ref. 13, 183, 229). Since such fibers would also be activated by electrical stimulation of

the nerves at group II strength, their central actions could confuse the overall picture. For example, these nonspindle group II fibers could project to a distinct set of inhibitory interneurons that might then make connections to homonymous extensor motoneurons. This possibility is incorporated in Figure 18 as the lower solid group II projection to the FRA interneurons. The central projections of secondary spindle afferents could then be quite separate and purely excitatory in nature.

The second hypothesis states that secondary ending projections may go to both excitatory and inhibitory interneurons (178). This hypothesis requires that excitatory interneurons be suppressed in the spinal and anesthetized cat (since inhibitory effects of electrical stimulation of muscle nerves predominate) and that excitatory interneuron activity predominate in the decerebrate preparation (in which excitatory effects predominate). This hypothesis necessarily implies that the net synaptic effects of afferent projections may be switched by descending pathways. Although no clear evidence of supraspinally mediated switching exists, there is now ample evidence of changing effects of peripheral stimulation during various phases of the locomotion cycle (see the chapter by Grillner in this *Handbook*), giving some credence to the general notion of switching.

One study (82) has reported the existence of inter-

neurons in the intermediate nucleus of the spinal cord that are activated selectively by electrical stimulation of muscle afferents at group II strength—selective activation was achieved by electrical blockade of group I afferent fibers. These results provide a possible neural substrate for polysynaptic group II effects. Nevertheless, the receptor origins of these fibers were not established and, as with the prospective Ib-interneurons, the nature and strength of any efferent synaptic connections remain to be determined.

#### *Projections From Groups III and IV Free Nerve Endings*

The receptor origins of muscle afferents with group III or IV fiber diameters are not completely certain; however, it is likely that these fibers originate in free nerve endings (264). Regardless of origin, the synaptic effects of these spinal projections usually conform with the patterns established by many other high-threshold afferents (124), hence their inclusion in the "FRA" category. The functional consequences of activating such afferents are also consistent with this classification, since thermal (120), chemical (79, 80, 209, 260, 161), and nociceptive or high-threshold mechanical stimulation of muscle belly or tendon (162) all activate muscle afferent fibers and give rise to potent flexion reflexes (230). Finally, as discussed in the next section, activation of these afferents could be responsible for the clasp-knife reflex in situations where the appropriate inhibitory interneurons are accessible to excitatory input from the groups III and IV afferents (see Fig. 18).

#### *Clasp-Knife Reflex*

The clasp-knife reflex is an abrupt collapse in force that occurs when a spastic limb is moved beyond some threshold angle. The functional contributions of various muscle afferents to the clasp-knife reflex have been reappraised in recent studies (33, 259). In each study, the clasp-knife reflex was examined using the decerebrate cat preparation in which there was introduced a dorsal hemisection of the thoracic spinal cord. This hemisection appears to interrupt descending dorsal reticulospinal fibers that inhibit interneurons of the flexor-reflex pathway releasing many segmental inhibitory reflexes (Fig. 19). Under these conditions stretch of the soleus muscle can be shown to induce a profound inhibition that depends jointly on muscle length (33, 259) and muscle force (259).

In the past the clasp-knife reflex has been attributed variously to the central actions of Golgi tendon organ afferents (11), to inhibition of fusimotor neurons (61), and more recently to central inhibitory effects of secondary spindle afferents (33). An examination of the responses of primary and secondary spindle and tendon organ afferent fibers during clasp-knife responses, however, failed to establish a correlation between the

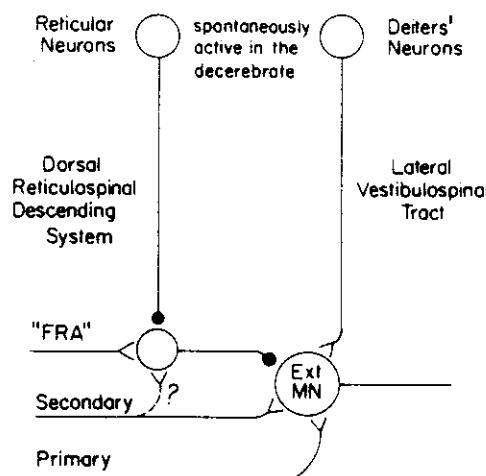


FIG. 19. Neural elements involved in clasp-knife reflex. Primary and secondary spindle afferents provide autogenetic excitation to extensor motoneurons. These neurons also receive tonic excitatory input from the vestibulospinal tract. In the decerebrate state, dorsal reticular neurons that inhibit segmental FRA interneurons are also active, reducing flexor-reflex activity. Section of the dorsolateral quadrants of the cord interrupts the dorsal reticulospinal pathway, thereby releasing segmental flexion reflexes. These include the clasp-knife reflex.

discharge of these muscle afferents and the quantitative features of the clasp-knife reflex (259). In addition a profound inhibition of soleus muscle force was induced by gentle mechanical stimulation of the muscle and tendon surface, at strengths insufficient to modulate primary or secondary afferent discharge to any significant degree. In sum, none of the established encapsulated receptors (spindle receptors and tendon organs) showed discharge patterns that were well correlated with the clasp-knife inhibition.

It has been known since the studies of Lloyd (172, 173) that electrical stimulation of groups III and IV muscle afferent fibers can induce inhibition of extensor motoneurons. It has also been determined that many groups III and IV fibers are activated by muscle stretch and contraction (80, 160). Moreover it is conceivable that a similar group of low-sensitivity muscle stretch receptors providing afferents with group II fiber diameter exists—in fact, several such group II fibers were isolated in the study of Rymer, Houk, and Crago (259). At the present time, the evidence with regard to stretch sensitivity of such afferents is quite fragmentary, and a more detailed study of nonspindle group II afferents is clearly warranted. In spite of the lack of direct evidence, on the basis of the joint length and force dependence of the clasp-knife reflex, the absence of detailed correlations between the clasp-knife characteristics and the discharge of identified spindle and tendon organ afferents, it seems likely that low-sensitivity stretch receptors (spanning groups II, III, and IV conduction velocity) may be responsible for the clasp-knife reflex. These low-sensitivity stretch receptors would presumably provide spinal projections

to inhibitory interneurons belonging to the "FRA" group. This possibility is included as the free nerve ending source of group II fibers in Figure 18. This approach, which is in accord with the first hypothesis of group II central effects (i.e., separate spindle and nonspindle group II pathways) does not eliminate the possibility of supraspinally mediated interneuronal switching; the need for a switching hypothesis simply becomes much less compelling.

The remaining issue concerns the reasons for the release of inhibitory reflexes (such as the clasp-knife reflex) in the spastic state. The relevant evidence has been provided by Burke et al. (33) and is summarized in Figure 19. These authors showed that section of the spinal dorsolateral fasciculus transformed rigidity of the decerebrate cat preparation into a state characterized by clasp-knife inhibition and other manifestations of spasticity. It was further argued that the spinal lesion interrupts the descending dorsal reticulospinal projection (60), which appears to suppress activity in flexor-reflex interneurons (65). Transection of these pathways would then release activity in previously inactive reflex circuits, including several that could potentially inhibit extensor motoneurons. A parallel between the animal model of spasticity and the human state was drawn by showing that excitatory cortical projections to dorsal reticulospinal neurons may be necessary to maintain activity in these pathways (10).

#### Long-Loop Reflexes

To this point we have assumed that the pathways mediating motor servo actions are located in regional segments of the spinal cord. However, this position is contrary to the views expressed in several influential reports dealing with stretch reflexes in intact subjects (67, 69, 185, 186, 206, 271). According to these and many other sources, the spinal component of the stretch reflex is both brief and small and is dominated by a long-loop reflex characterized by medium-latency electromyographic responses to limb perturbations.

The evidence in support of long-loop contributions is derived partly from the patterns of electromyographic (EMG) response to limb perturbations in human and monkey preparations and partly from recordings in motor cortex and related structures in both anesthetized and conscious monkeys. With regard to the EMG responses, investigators in several laboratories have found that abrupt muscle stretch provokes a peaked pattern in the processed EMG, which has subsequently been characterized in terms of grouped discharges of motor units (17). The particular pattern obtained varies somewhat between laboratories, but in each case it consists of several short- and intermediate-latency peaks in the averaged rectified electromyogram. For example, Tatton and his collaborators have recognized the four peaks shown in Figure 20, the first of which (M1) begins at a latency consistent with an origin in the Ia monosynaptic path-

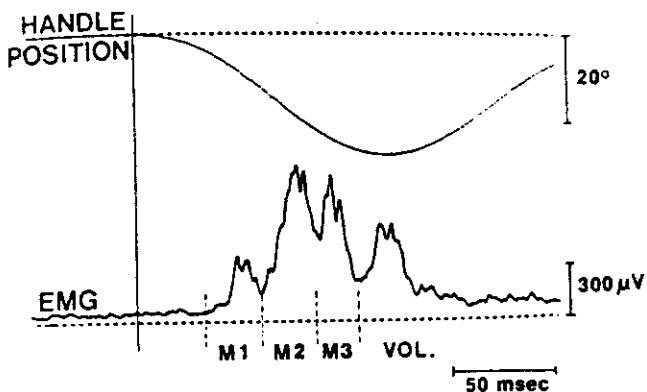


FIG. 20. Averaged rectified EMG response from wrist extensors of a normal subject following sudden flexor displacements of the wrist. The vertical line indicates when torque motor was turned on. Subject was instructed to actively return the handle to the central zero as soon as the displacement occurred. Onset latencies for the components of the response: M<sub>1</sub> = 32 ms; M<sub>2</sub> = 59 ms; M<sub>3</sub> = 85 ms; "voluntary" activity = 107 ms. Downward deflection of handle position trace represents flexion at the wrist. [From Lee and Tatton (169).]

way and the last of which is attributed to the onset of a voluntary response. The other two peaks (M<sub>2</sub> and M<sub>3</sub>) sometimes merge into a single response that appears to be similar to what other investigators have called the medium-latency component (5, 164) or the functional stretch reflex (206). The A- and B-waves described by Marsden and collaborators (186) may be analogous to the later half of Tatton's M<sub>1</sub> component and his M<sub>2</sub> component, respectively (190).

The common theme in all of this is the identification of medium-latency components in the EMG responses to abrupt stretch, components that follow the monosynaptic tendon-jerk response but occur before the presumed latency of a voluntary response. These medium-latency responses have been attributed to conduction through excitatory long-loop pathways between the segment and supraspinal structures because of the apparent correspondence between the timing of EMG response components and the calculated times required to traverse such (hypothetical) long-loop pathways. This emphasis on latency derived initially from a study of human stretch reflexes by Hammond (114) showing that medium-latency (50–60 ms) EMG components of response to biceps stretch are modifiable by prior instruction. Because of their delayed onset and their modifiability, these responses were assigned to long-loop pathways traversing supraspinal structures.

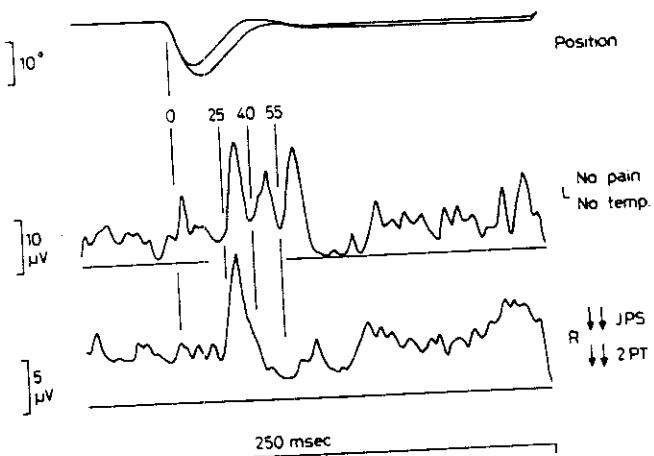
The notion of long-loop connections was subsequently reinforced and expanded by Phillips (236, 237) and Heath et al. (118), who described neurons in cortical area 3a that were responsive to activation of Ia-afferents. It was further suggested that these neurons acted as part of the afferent limb of a transcortical servo loop, the efferent limb being provided by the corticospinal projection. Recordings from motor cor-

tex (42, 67, 70) provided evidence for such afferent connections and revealed that pyramidal tract neurons responded at short latency to limb perturbations that would certainly have activated spindle afferents. More direct evidence regarding the specific contributions of spindle afferents to the activation of corticospinal neurons in conscious primates has been provided recently by studies in which selective muscle stretch was induced without activating cutaneous afferents (290). In addition, electrophysiological evidence, derived from the cat, has supported the existence of connections between areas 3a and 4 (292) although a more direct pathway, from the ventralis posterior lateralis pars oralis (VPLo) nucleus of the thalamus appears to be the source of muscle afferent projections to area 4 in the monkey (8). Also it has been reported recently that pyramidal neurons responding to limb perturbation project back to the same muscles that are activated by limb perturbations (38).

The above results provide strong evidence for the existence of a transcortical loop. However, it remains unclear whether this pathway functions as a servo-regulatory loop that should be included within the functional boundaries of the motor servo, or whether its main function is in the genesis of reaction-time movements [see Evarts and Vaughan (71) and Houk (128) for alternative views]. Furthermore if it does function as a motor servo loop, one would like to know what this pathway adds to the servoregulatory actions mediated by the segmental pathways discussed earlier.

Many investigators have discounted the possible contributions of segmental pathways to the medium-latency responses on the basis of the following arguments. First, when different muscles are compared, the latency of these EMG components was found to increase in proportion to the distance of the muscle from cortex (187). Second, it was claimed that medium-latency EMG waves were modifiable by prior instruction (67–71, 114, 191). Third, their magnitude was influenced selectively by lesions of the dorsal columns (168, 188) and sensorimotor cortex (2, 189). An example of a reduced intermediate-latency EMG response, taken from a subject with a unilateral brain stem lesion, is shown in Figure 21.

Although a transcortical loop could certainly provide delayed excitation to spinal motoneurons, it might not act in the production of the peaked pattern of EMG. The medium-latency EMG waves could equally arise from purely segmental mechanisms, such as strong synchronization of motor unit discharge, synchronization of afferent input, fluctuating inhibition, conduction through polysynaptic spinal pathways, or from oscillatory behavior of spinal neuronal networks (but cf. ref. 17). In fact, medium-latency peaks are seen in the EMG responses of decerebrate and spinal cats and monkeys (83, 213, 226, 273), all of which lack the transcortical loop. The close resemblance between the EMG components seen in comparisons of intact and spinal animals (83) appears to



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FIG. 21. Response to a fast brief stretch of the long thumb flexor in a patient with a lesion in the right brain stem (due to stroke), causing loss of pain and temperature sensation in the left arm and loss of appreciation of joint position, vibration, and tactile discrimination in the right arm, but no apparent motor deficit. The upper records show the angular position of the right and left thumbs. The middle and lower records show the full-wave rectified EMG recorded from flexor pollicis longus of the left and right hands. Subject held the thumb stationary against a standing force of 2 N. At time 0, indicated by the vertical marker, a force of 30 N was applied for 3 ms. Each trace is the average of 24 trials. In the EMG record from the left long thumb flexor there are clear responses at monosynaptic latency (25 ms), and later responses at 40 and 55 ms. In the record for the right long thumb flexor, the monosynaptic response is still apparent, but the medium- and long-latency responses are not apparent. [From Marsden et al. (190).]

demonstrate that the segmentation of EMG responses into short-, medium-, and long-latency components is, in fact, attributable to spinal mechanisms. The finding does not exclude the possibility that the amplitudes of these waves are modulated by transcortical and other supraspinal loops. The previous findings of a disappearance of specific medium-latency components subsequent to various brain lesions appear to be attributable mainly to alterations in the central excitatory state of the spinal cord (213). In *Effect of Instructional Set*, p. 294, we review arguments suggesting that the supraspinal modulation of medium-latency EMG components, when present, has more to do with the generation of reaction-time movements than with motor servo actions.

#### Summary

The major autogenetic connections are summarized in Figure 18, which also reinforces the point that of the autogenetic reflex connections of muscle afferents (the solid lines) the monosynaptic are the best studied. This emphasis on monosynaptic connection is certainly an outcome of the technical difficulties associated with the study of interneurons and polysynaptic connections and has no bearing on the functional significance of interneuronal connections. The evidence supporting long-loop connections as compo-

nents of the motor servo is reviewed and shown to be inconclusive at best.

#### SIMPLIFIED ANIMAL MODELS

A normal or near normal central excitatory state is one of the key requirements for productive studies of length and force control mechanisms. In other words motoneurons and interneurons in reflex pathways must receive appropriate tonic levels of background excitation and inhibition, since an absence or modification of this activity results in abnormal depression or an uncharacteristic enhancement of reflex transmission. A normal excitatory state is insured in studies of intact subjects, but the responses obtained may represent a complex hierarchy of control actions. For example, presumed motor servo actions may be contaminated by reaction-time movements, as discussed in *Effect of Instructional Set*, p. 294. Furthermore surgical procedures that simplify the experimental system are nearly always required to identify the afferent sources and specific neuronal pathways that mediate the observed responses. Because of these conflicting requirements, we must judiciously synthesize the results from intact subjects and from subjects in which transections and ablations have been used to create simplified experimental systems. This section reviews the characteristic features of two simplified systems that have been particularly useful in studies of the motor servo.

#### Decerebrate Preparation

The discovery of many basic motor reflexes and the analysis of their neural mechanisms has depended critically on several fortunate properties of the decerebrate state. This state of the spinal cord and lower brain stem is produced by a transection through the mesencephalon (263) or by an interruption of the cerebral circulation (239). Upon recovery from the anesthetic, a typical decerebrate animal makes few spontaneous movements (other than breathing and sometimes stepping) yet is highly responsive to a variety of natural stimuli. The responses thus provoked are simple motor patterns that appear to represent fragments of the more complex behavioral acts of the intact animal, some of which are reestablished if the animal is maintained for the long term (12). The decerebrate animal usually shows a depressed responsiveness to nociceptive stimuli that allows the experimenter to undertake surgical maneuvers (limb dissection, laminectomy) essential in the analysis of mechanism, without greatly disrupting the reflexes under investigation. Another useful feature is the tonic activity of the extensor motoneurons (extensor rigidity), which mimics tonicity during standing. These combined features made possible the discovery and analysis of the stretch reflex by Liddell and Sherrington in 1924 (170).

The characteristic loss of spontaneous movements in the decerebrate is the combined result of an interruption of neural circuits through the brain above the lesion and a depression of neural circuits, such as the spinal locomotor network (see the chapter by Grillner in this *Handbook*) and brain stem postural network (see the chapter by Wilson and Peterson in this *Handbook*), below the lesion. The depression of nociceptive reflexes is attributable, at least in part, to a tonic inhibition of the flexor-reflex pathway mediated by enhanced tonic discharge conducted in the dorsal reticulospinal descending system (65). This tonic inhibition is thought to contribute to extensor rigidity by blocking inhibitory transmission from the FRA to extensor motoneurons [cf. Grillner (102); Fig. 19].

Many of the properties of the released extensor tonus are the same as those associated with normal postural contractions. In both cases the recruitment of motor units follows the size principle [(119); see the chapter by Henneman and Mendell in this *Handbook*]. Also the tonic discharge of skeleotomotor neurons depends partly on excitatory input from spindle receptors that derives from tonic activity in fusimotor neurons and partly on direct input to  $\alpha$ -motoneurons (201, 262, 283). In the decerebrate a major source of the combined  $\alpha$ - $\gamma$ -input descends in the vestibulospinal pathway (cf. ref. 105), presumably mimicking a postural motor command. This activity results from spontaneous discharge of Deiters' neurons (Fig. 19).

The levels of tonus are independent of potential variations in the gain in each of the autogenetic reflex pathways. An enhanced sensitivity to tendon taps (hyperreflexia) is associated with the decerebrate state, but this measure does not adequately assess gain. Hyperreflexia is caused by a general increase in motoneuron excitability that probably reflects changes in bias rather than authentic modifications in gain. For example, if the motoneuron pool is biased closer to threshold, the same gain in the Ia pathway will result in a larger motor volley and tendon jerk.

The gain of length versus force feedback (138) and the relative gain in primary and secondary spindle receptor pathways (197) have been estimated in the decerebrate cat, but there are no comparable measures available for intact subjects. There is qualitative indication (48), however, that the gain of force feedback via tendon organs is probably higher in human subjects than in the decerebrate cat. It is also known (59, 126) that IPSPs mediated by Ib volleys are depressed in the decerebrate cat, in contrast with the spinal cat, but no comparable measures in intact subjects are available.

#### *Spinal Preparation*

Acutely spinalized animals lack the requisite excitatory state of interneuronal pathways for well-developed stretch reflexes due to the interruption of tonic descending activity caused by the transection (37,

203). For example, it is apparent from Figure 19 that a spinal transection would interrupt tonic excitation from Deiters' neurons and would also release the inhibition of extensor motoneurons due to any spontaneous activity in the flexor-reflex pathway. Recovery from this depressed state (spinal shock) is slow and incomplete. Although deep tendon reflexes may return after a period of minutes to hours, they represent only a partial manifestation of authentic stretch reflexes, a portion mediated predominantly by the monosynaptic Ia pathway. In spite of this, profitable studies of stretch reflexes have been conducted on chronically maintained spinal animals (234) and on acute spinal animals after the administration of certain pharmacological agents that reverse the depressed excitability associated with spinal shock. Agents that have been used successfully are 5-HTP [5-hydroxytryptophan; (4)] and L-dopa [L-dihydroxyphenylalanine; (88)]. Since these substances are precursors of transmitters in descending fibers but not in fibers of intrinsic spinal neurons or of dorsal root afferents, one can reasonably assume that their actions substitute (partially) for the removal of tonic descending activity caused by the cord transection.

The importance of the experiments with spinal animals is that they demonstrate the sufficiency of segmental pathways in the mediation of motor servo actions. But for convenience, most of the studies on functionally isolated muscles have been conducted in the decerebrate animal, under the likely assumption that the responses obtained are also mediated by segmental reflex pathways.

#### *Summary*

The decerebrate animal appears to be a good preparation for the study of motor servo function and mechanism. The extensor rigidity of this preparation may suffer from a depressed gain in the Ib pathway, but it has a normal order of motor unit recruitment and a coactivity of  $\alpha$ - and  $\gamma$ -motoneurons that is one of the characteristic features of motor servo activity in intact subjects.

#### TONIC STRETCH REFLEX IN FUNCTIONALLY ISOLATED MUSCLES

##### *Basic Features of the Stretch Reflex*

Liddell and Sherrington (170) and Denny-Brown (49) developed the basic procedures still used today for studying the stretch reflex of functionally isolated muscles in decerebrate and spinal animals. An individual muscle, usually an extensor since rigidity is best developed in extensors, is functionally isolated by transecting all (or most) of the cutaneous and motor nerves in the experimental limb, sparing the nerve to the muscle under study. This leaves intact the autogenetic reflex pathways to and from the muscle. The muscle

is then attached to a mechanical or electromechanical stretching device that is fitted with transducers for measuring the force and length of the muscle and sometimes velocity and acceleration. Usually muscle length is controlled, thus serving as the input variable, while force is monitored as the output variable. Electromyographic (EMG) activity can also be monitored to provide a measure of motor output. The system thus available for experimental study is that portion of the motor servo enclosed by dashed lines in Figure 1.

The outstanding property of this neuromuscular system is a graded resistance to length change that to a first approximation resembles the properties of a spring. Figure 22, which reproduces one of the original records from Liddell and Sherrington (170), illustrates this and other characteristic features of the stretch reflex. Throughout the 1-s period during which the stretch was applied (dotted trace labeled T), the developed force (trace M) increased in a springlike manner approximately in proportion to the amount of stretch. However, unlike the behavior of a purely elastic system, force decayed slightly while the muscle was held at the longer length. The maintained component of force in response to a maintained stretch is called the tonic stretch reflex.

The degree of adaptation (i.e., the amount of decay in force) depends on several factors. In the first place it varies considerably between preparations and becomes greatest (adaptation to zero active force) in animals that for other reasons are judged to be in poor condition. In addition, however, nearly all preparations show some degree of adaptation, which is simply an expression of dynamic properties analogous to those of a mechanical system consisting of a spring plus a dashpot or some other frictional element in parallel. Thus, if the muscle is stretched to the same

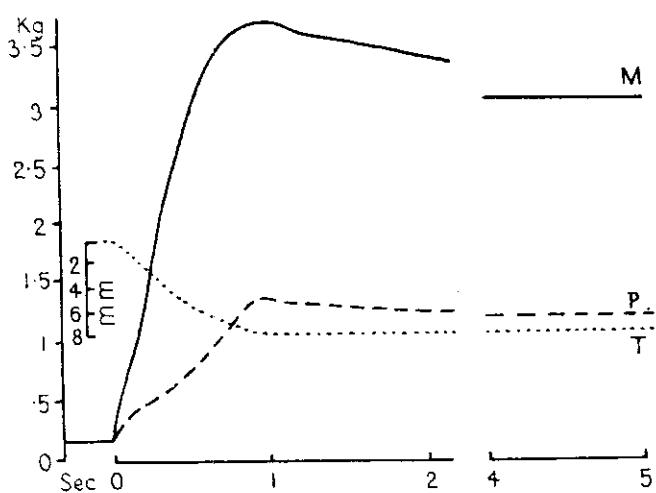


FIG. 22. Stretch reflex of quadriceps muscle of decerebrate cat. Trace T shows the time course of stretch, M shows the force produced in the muscle with autogenic reflexes intact, and P shows the passive muscle force produced after cutting the motor nerve. [From Liddell and Sherrington (170).]

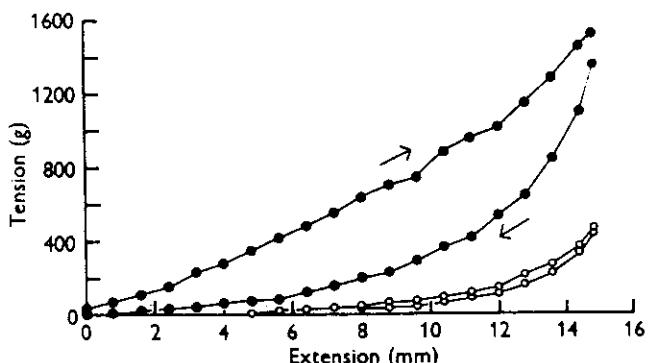


FIG. 23. Stretch reflex of the soleus muscle of decerebrate cat. Upper curve shows total tension during stretch at 1.7 mm/s. The stretch was maintained for 2 s before shortening muscle at the same velocity to obtain the descending curve. Lower curves show the passive tension obtained during stretch and release while the reflex was being inhibited. [From Matthews (193).]

length more rapidly, force rises more rapidly and to a higher value during stretch and correspondingly decays more at stretch plateau. Force typically decays to the same steady-state value independent of stretch velocity, as an expression of the static (or purely elastic) properties. Dynamic features of stretch reflexes will be given more detailed treatment in DYNAMIC RESPONSES TO MECHANICAL DISTURBANCES, p. 297.

The remaining trace in Figure 22 (the dashed trace labeled P) represents the passive component of the total force M. Liddell and Sherrington obtained it by applying the same stretch to the muscle after its nerve had been cut, and they interpreted the difference as a demonstration that a major component of the observed resistance is of reflex origin. Note that according to conventional terminology the term stretch reflex applies to the entire stretch response, even though its passive component is quite unrelated to reflex function. The difference between total force and its passive component is called the active component of the stretch reflex, which can be further broken down into an active muscle mechanical component and a neurally mediated component, as will be elaborated in Mechanically and Neurally Mediated Components, p. 285.

#### Static Force-Length Relations

A useful way of displaying the static properties of the motor servo is on a plot of force versus muscle length as illustrated in Figure 23, which is taken from an early study by Matthews (193). The muscle was stretched at a low velocity ( $\rightarrow$ ) to maximal physiological length, held at that length for a couple of seconds, and then shortened at the same low velocity ( $\leftarrow$ ). The closed points represent the total force, whereas the open points represent the passive forces observed during a stretch-release sequence applied to an inactive muscle. The fact that force (tension in Fig. 23) decayed slightly when the muscle was held at maximal length

and decreased along a lower curve when the muscle was shortened is again indicative of a dynamic component, in spite of the fact that the velocity was extremely slow (the stretch phase occurred over a 15-s time period). Although Figure 23 thus does not show the true static relation between force and length, one can imagine it as a curve located somewhere between the two curves showing response during slow stretch and slow release. In order to approximate more closely the truly static force-length relation, different amplitudes of stretch have been applied and maintained for 4–30 s (97, 225), at which time force has decayed to nearly its steady-state value. The heavy solid curve in Figure 24 shows a static relation obtained by Nichols (224) in a decerebrate cat that demonstrated strong rigidity.

Static force-length relations are usually approximately linear, although there may be a concave region near threshold and either saturation or upward curvature at longer lengths. Assuming an approximately linear relation, the slope of a straight line fit to the data will represent the static stiffness of the motor servo

$$f = K(x - x_0) \quad (22)$$

where  $f$  and  $x$  are muscle force and length, respectively,  $x_0$  is the threshold of the stretch reflex, and  $K$  is the static stiffness. Equation 22 is a static, linear approximation to the more general expression for force-length relations considered earlier (Eq. 16).

For the soleus muscle of the cat, which has been most extensively studied, static stiffness may be as high as 3 N/mm (Newtons per millimeter; 1 N/mm is equivalent to 100 g·wt/mm). At the other extreme there may be essentially no static resistance to stretch. While zero stiffness is considered to be a sign of poor preparation, values in the range 0.2 to 0.5 N/mm are not uncommon (97, 150, 193). In contrast, the static stiffness of the cat's medial gastrocnemius is typically 2–3 N/mm and can be as high as 5 N/mm (45), but the maximal force of this muscle is approximately 4 times greater than that of the soleus.

#### Normalized Stiffness

If values of static stiffness are to be compared between species or between different muscles in the same species, one should convert them to some normalized measure. Here it seems reasonable to express the force and length changes used to compute the slope of the force-length relation as fractions of the normal physiological ranges

$$K_n = \frac{\text{force change}}{\text{force range}} / \frac{\text{length change}}{\text{length range}} \quad (23)$$

or

$$K_n = K \cdot \frac{\text{length range}}{\text{force range}} \quad (23a)$$

where  $K_n$  represents the normalized stiffness; a value of 1 would signify that stretching a muscle over its full range would result in a force modulation over its full range. For a cat soleus muscle the force range is 20–25 N and the length range is 25–30 mm. Thus, normalized stiffness ranges from 0.2 to 3 and a typical value in an animal with well-developed rigidity is in the vicinity of 1. For the cat gastrocnemius the force range is about 4 times greater whereas the length range is similar. This together with the absolute stiffness values given earlier yields a normalized stiffness in the vicinity of 0.7, only slightly less than that for soleus.

#### Mechanically and Neurally Mediated Components

The force-length curve of a tonic stretch reflex (Fig. 23) and the active length-tension curve of a muscle (Figs. 14 and 15) are similar in that both represent a springlike property that opposes stretch (97, 103, 193, 231). In addition, the slopes of the curves, which represent static stiffness, can be remarkably similar under certain selected conditions. Detailed comparisons using the soleus muscle in the decerebrate cat indicate that the stiffness of a typical tonic stretch reflex is approximately matched by the slope of the steepest portion of the length-tension curve of the stimulated muscle (103, 107, 225). In normalized units, the static stiffness of a fully recruited muscle can be as large as 2, although normalized stiffness is clearly less than this at nonoptimal initial lengths and stimulus rates (Fig. 15), and it decreases at lower levels of recruitment (Eq. 20). It seems clear that autogenetic reflexes do not greatly augment the stiffness of the motor servo over and above the stiffness that could be achieved by simply recruiting all of the motor units of a muscle.

It has also been shown (107, 240) that a level of spinal motor output capable of producing this maximal or near maximal muscular stiffness can occur spontaneously after the dorsal roots have been sectioned to eliminate the normal excitatory input from muscle spindles. The production of this high level of motor output without reflex involvement requires a strong central excitatory drive to the relevant motoneurons. Pompeiano (240) obtained the requisite excitation in forelimb motoneurons by combining a thoracic spinal transection to eliminate ascending inhibition with a traditional decerebration to produce descending excitation. Grillner and Udo (107) obtained it in lumbosacral motoneurons by decerebellation of decerebrate animals or by administering the excitatory pharmaceutical agent 5-HTP to spinal animals.

The former results demonstrate that a major portion of motor servo stiffness could be (but is not necessarily) of muscle mechanical, rather than neural, origin. However, electromyographic (EMG) recordings demonstrate that stretch and release do result in a prominent reflex modulation of motor output. This EMG response is monotonically related to muscle length and displays a distinct threshold (49, 97). The

existence of a threshold value of muscle length below which the motoneuronal pool is quiescent suggests that rather different mechanisms are responsible for the production of motor servo force-length relations and muscle mechanical length-tension curves, even though the two often have the same exterior appearance. While muscle length-tension curves mimic motor servo curves by displaying an apparent threshold length below which the developed force is nearly zero (Fig. 15), this results from mechanical limitations rather than from the motoneuronal quiescence that characterizes actual motor servo performance. The modulation of the EMG observed above threshold is also contrary to a simple mechanical explanation, although the quantitative significance of this modulation requires evaluation.

Grillner and Udo (108) reported that a disproportionately large number of soleus motor units in the decerebrate are recruited during the first few millimeters of slow stretch beyond threshold, which led them to suggest that essentially the whole motor pool is recruited near threshold and muscle length-tension curves account for the stretch reflex beyond this point. An alternative interpretation of their data follows from the size principle (see the chapters by Burke and by Henneman and Mendell in this *Handbook*). The large number of units recruited near threshold are probably small ones, whereas the few recruited at longer lengths are probably large. The recruitment of a few large units at longer lengths could easily produce as much resistance as the recruitment of many small units of shorter lengths. The equation

$$f = F \left( \frac{n}{N} \right)^2 \quad (24)$$

provides a quantitative expression for this phenomenon, called the recruitment nonlinearity, that was derived empirically from published motor unit data (138);  $f$  and  $n$  are force and the number of active motor units whereas  $F$  and  $N$  represent the maximal values of these variables. According to this expression, half of the motor units in the soleus pool should be recruited when the force is only 25% of maximum, which appears to be in accord with Grillner and Udo's (108) observations. A similar nonlinear relation between force and number of motor units applies to the medial gastrocnemius muscle (39) and to the triceps surae as a whole (see the chapter by Burke in this *Handbook*).

The difficult problem of deducing mechanical efficacy from electrical events can be circumvented if muscular and neural components are estimated myographically. Figure 24 shows an example of the results of a myographic analysis by Nichols (224), made possible by Grillner and Udo's (107) finding that all tonically active soleus motor units discharge at approximately 8 impulses/s in the decerebrate preparation. The heavy solid curve shows the static force-length relation of the intact motor servo, and the broken

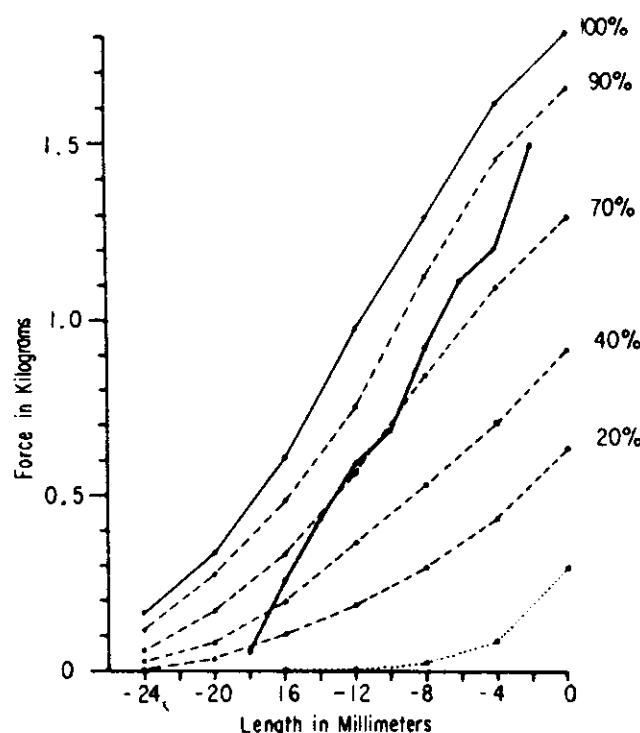


FIG. 24. Comparison of tonic stretch reflex with length-tension curves for the soleus muscle of decerebrate cat. The *heavy line* represents the static force-length relationship obtained by waiting 30 s for adaptation at each successive length. Other *curves* are length-tension curves obtained at the end of the experiment by stimulating different proportions of the cut ventral roots at 8 pulses/s. The *passive curve* is *dotted*. [From Nichols (224).]

curves show the 8 impulses/s length-tension curves of the same muscle at different percentages of artificial recruitment. The mechanical component of the overall reflex can thus be gauged by comparing slopes at each point of intersection. It is apparent that mechanical stiffness was small near threshold and grew progressively to become approximately 50% of motor servo stiffness at the longest length studied, at which point approximately 90% of the muscle mass was recruited.

The results discussed in the previous paragraph support the proposal for a greater rate of motor unit recruitment near threshold. The example also shows that recruitment continues to be an important mechanism throughout the normal range of the reflex. What seems particularly remarkable is the relative constancy of overall reflex stiffness (the nearly constant slope of the heavy curve) as the origin of this property shifts gradually from a neural to a mechanical basis.

#### *Actions of Control Signals on the Motor Servo*

Electrophysiological studies have shown that pathways descending from the brain to the spinal cord have direct actions on  $\alpha$ - and  $\gamma$ -motoneurons, as well as actions on interneurons in reflex pathways and on

presynaptic terminals [cf. ref. (177)]. It is sometimes presumed that the interneuronal and presynaptic actions are particularly important in controlling reflex responsiveness and that movements, instead, are controlled by direct actions on motoneurons. Certainly, there is now good evidence that movement commands are sent to both skeleto-motor and fusimotor neurons (99);  $\alpha$ - $\gamma$ -Relations, p. 308.] and it is also clear that reflex transmission, as tested by electrophysiological techniques, can be altered by descending activity (see the chapter by Baldissera, Hultborn, and Illert in this *Handbook*). Changes in reflex transmission, however, only demonstrate the anatomical convergence of descending and reflex pathways; the normal actions of a descending input that converges on a reflex pathway might add to an ongoing input from the periphery and thus bias reflex threshold, rather than alter the gain of the reflex.

Studies in decerebrate animals in which control signals to the motor servo (cf. Fig. 1) are altered in a variety of ways have in most instances demonstrated a simple shift in the threshold of the force-length relation shown by the dashed curve in Figure 11, corresponding to a bias type of input, rather than a change in its slope as illustrated by the dotted curve in Figure 11, corresponding to gain control. Matthews (193) reported on the effects of activating synergistic, antagonistic, and crossed-extensor reflex pathways, and on the consequences of spontaneous variations in rigidity and the depression caused by a procaine paralysis of  $\gamma$ -fibers. The salient effect of each of these procedures was a shift of the force-length relation along the abscissa, i.e., an alteration in stretch reflex threshold with little change in the shape of the curve. Excitatory inputs lowered the threshold, whereas inhibitory inputs moved it to a longer muscle length. As shown in Figure 25, Feldman and Orlovsky (76) obtained the same result with tonic stimulation of descending pathways. Various excitatory and inhibitory combinations obtained by stimulating ipsilateral and contralateral Deiters' nucleus, the pyramidal tract, and the medial medullary reticular formation resulted simply in threshold changes. However, Kim and Partridge (157) reported that utricular nerve stimulation sometimes caused changes in shape (at low levels of central excitatory state) as well as shifts in the threshold. Even in these cases, the family of stretch-reflex curves was nonintersecting, which suggested that there was only one degree of freedom in the modulatory effect. In other words, there was no evidence that force and slope could be independently controlled.

The basic uniformity of these results is striking in view of the variable  $\alpha$ - to  $\gamma$ -motoneuron contribution to the different simulated motor commands. Descending and crossed-extensor pathways are known to coactivate  $\alpha$ - and  $\gamma$ -motoneurons, the synergistic and antagonistic pathways activate mainly  $\alpha$ -motoneurons, and paralysis of the  $\gamma$ -motoneurons alters only the  $\gamma$

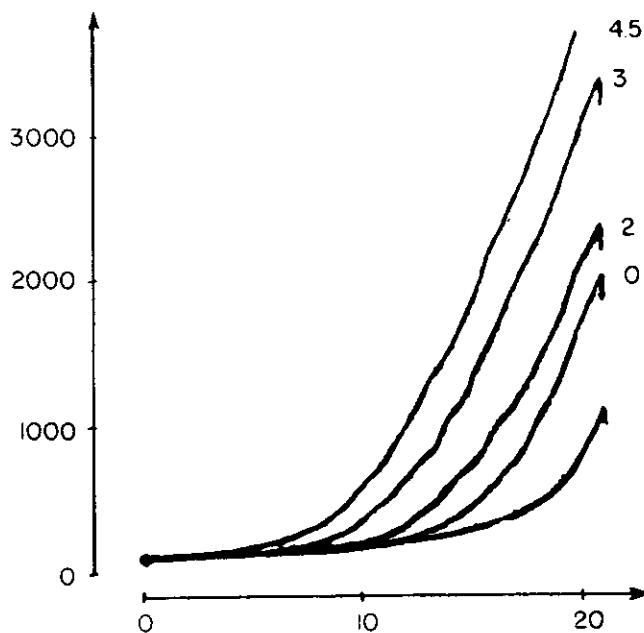


FIG. 25. Family of force-length relations obtained at different intensities of Deiters' nucleus stimulation. The ordinate is the force in g.wt and the abscissa is muscle extension in mm. Reflex force was registered while the muscle (gastrocnemius in decerebrate cat) was stretched slowly to its maximal physiological length. Deiters' nucleus stimulation at a fixed rate was delivered before and throughout the period of stretching, to mimic a constant level of central motor command. The numbers represent stimulus intensity (V) and the unlabeled curve is the response of the passive muscle. [From Feldman and Orlovsky (76).]

signal. Apparently the static performance of the motor servo is rather independent of the  $\alpha$ - $\gamma$ -ratio.

More profound alterations in the slope or general shape of the force-length relation have also been described. Changes of this type sometimes characterize spontaneous variations in rigidity (97, 225), and they are regularly produced by spinal lesions of the dorsolateral columns that, as discussed earlier, convert a normal stretch reflex into a clasp-knife reflex. Such findings must be interpreted with caution, since they could be simply the result of pathological phenomena and play no role in normal motor servo function. This issue is better addressed in experiments with intact subjects (cf. subsection *Gain Variation vs. Gain Control*, p. 296).

#### *Dependence of Incremental Stiffness on Initial Force*

Often it is not feasible or practical to obtain complete force-length relations. Thus many investigators have studied the motor servo in terms of incremental responses to changes in muscle length applied about some initial length and initial level of reflex force. If the length changes are applied at a low velocity, or if they are maintained until a steady state is reached, the ratio of force change to length change provides a

measure of static stiffness that can be compared with the slope of the static force-length relation, in analogy with the earlier discussion of incremental muscular stiffness.

It is apparent from an inspection of a family of typical force-length curves (Fig. 25) that the slope (representing stiffness) is not constant. Since the curves are generally nonintersecting, specification of an initial force and muscle length would be expected to define quite adequately the initial operating conditions for an incremental analysis. Furthermore the shapes of the different force-length curves in a family are quite similar, as if a single curve were simply translated along the abscissa. Correspondingly, one would expect that most of the variation in the stiffness determined from incremental measurements would be dependent on the initial force, with much less dependence on initial length. These expectations are well supported by a variety of studies with functionally isolated muscles (20, 76, 122, 138, 225, 226, 254). Typically one finds that incremental stiffness increases at low initial forces, flattens to become independent of initial force throughout a midrange, and may decrease once more at very high initial forces. This dependence on initial force is illustrated by the individual curves in Figure 26 for observations at each of several initial lengths. The basic similarity of the curves illustrates an absence of any major dependence on initial length.

Figure 27 compares several incremental stretch responses to illustrate the dependence of the reflex and its underlying mechanical component on the initial force. The mechanical component was small at low initial forces, due to the fact that only a few fibers were active, but with recruitment it grew to become approximately 3 N/mm, which represents essentially 100% of the total quasistatic reflex response estimated 1 s after ramp onset. In contrast with the simple proportionality between the mechanical component and the initial force, overall reflex stiffness rose steeply from a zero value when the reflex was modulated below threshold (not shown) to a high value at relatively low forces. It then remained stable at about 3 N/mm throughout the range illustrated, due to the fact that the neural component (indicated by the difference between R and M traces) decreased as the muscular component increased. The changes associated with the dynamic phase of the response are also intriguing, but discussion will be postponed until DYNAMIC RESPONSES TO MECHANICAL DISTURBANCES, p. 297.

#### Loop Gain of Force Feedback

The material presented to this point suggests that the contribution of muscle mechanical stiffness to the stretch reflex can be either large or small; it varies considerably depending on the initial conditions. Autogenetic reflexes appear to produce compensatory responses that reduce the dependence of overall reflex stiffness on initial muscle length and force. Although

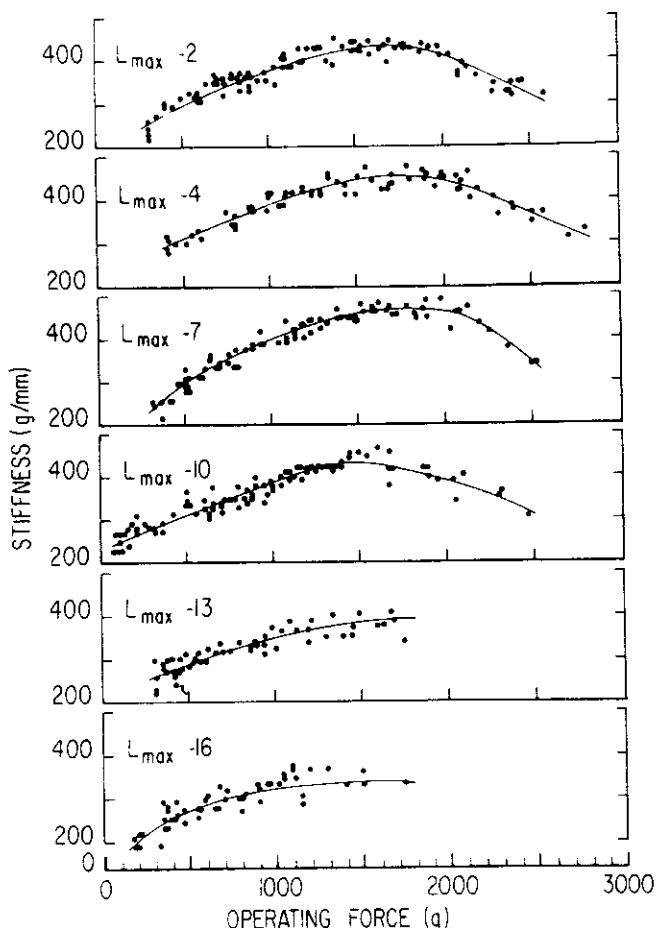


FIG. 26. Incremental stiffness of the stretch reflex in the decerebrate cat as a function of initial force and initial length. At each initial length the initial (or operating) force was modulated by eliciting a strong reflex, and stiffness was measured from responses to 1-mm pulses lasting 200 ms that were applied to the soleus muscle as force progressively decayed. Each point represents the incremental force-to-length ratio at the end of one pulse. [From Hoffer and Andreassen (122).]

these results support the hypothesis for stiffness regulation, they provide no information concerning the respective roles of spindle receptor and tendon organ pathways. A useful initial approach to this problem of partitioning regulatory actions between length and force-feedback pathways was developed by Houk and collaborators (138) on the basis of a linear analysis of the system. The simplified version of this analysis given here ignores the effects of tendon elasticity, although comment concerning these effects is made at an appropriate point.

An equation giving motor servo stiffness in terms of the three underlying components illustrated earlier in Figure 10 can be derived by substituting the expression for  $\Delta e$  in Equation 11 into Equation 13

$$\Delta f = K\Delta x + Ag_s\Delta x - Ag_T\Delta f \quad (25)$$

$$\frac{\Delta f}{\Delta x} = \frac{K + Ag_s}{1 + Ag_T} \quad (26)$$

where  $K$  is the mechanical stiffness of the muscle,  $Ags$  is the component of stiffness generated by length feedback from spindle receptors, and  $Ag_T$  is the dimensionless loop gain of force feedback. (The inclusion of series elasticity results in an additional component of force feedback produced by unloading of spindle receptors during contraction.) The attenuation factor  $1/(1 + Ag_T)$  represents the inhibitory force-feedback component as shown in Figure 10.

Values of loop gain appreciably  $<1$  clearly have no effect on overall stiffness ( $\Delta f/\Delta x$ ), and it can also be shown that this is the condition for no beneficial effect aiding stiffness regulation. Assuming that  $Ag_T$  is  $\ll 1$ , Equation 26 can be rewritten as

$$\frac{\Delta f}{\Delta x} = K + Ags \quad (27)$$

Thus with low loop gains, stiffness will simply be the sum of a mechanical component and a spindle feedback component.

At the other extreme, if the loop gain of force feedback were  $\gg 1$ , Equation 26 could be written

$$\frac{\Delta f}{\Delta x} = \frac{K}{Ag_T} + \frac{g_s}{g_T} \quad (28)$$

Large values of loop gain would progressively reduce the dependence of motor servo stiffness on the mechanical stiffness ( $K/Ag_T$  would go to 0), leaving stiffness to be determined by the ratio of the gains in  $g_s/g_T$  and tendon organ pathways. This diminution of dependence on variations in mechanical stiffness could be an important mechanism for stiffness regulation. In view of these potential advantages, it is disappointing that measurements of the loop gain of force feedback in functionally isolated muscles have yielded rather low values.

Measurement of the loop gain of force feedback requires some way of injecting a disturbance within the force-feedback loop that goes from motoneurons to muscle to tendon organs back to motoneurons. It is also important to minimize spindle feedback effects either by keeping the muscle isometric or by using the same stretch amplitude for test and control observations. Houk and collaborators (138) held the cat soleus muscle isometric and studied the tendon organ pathway by stimulating small ventral root filaments to produce disturbance forces within the muscle. Loop gain, gauged from the extent of attenuation of the internal disturbance, was never greater than 1 and a typical value was 0.5. In a similar series of experiments Jack and Roberts (147) found values in the range 0–0.15. Aymer and Hasan (256) used dantrolene sodium to produce changes in soleus contractility that acted similarly as internal disturbances. The incremental EMG response to a standard test stretch was not significantly influenced by drug treatments that decreased contractility by 20%–65%, indicating that loop gain was negligibly small. Finally, Hoffer and Andreas-

sen (122a) have used a stimulation-induced potentiation of soleus muscle contractility as an internal disturbance. They reported no appreciable attenuation of the potentiation by reflex action, which again suggests that loop gain is negligibly small in the functionally isolated soleus muscle of the decerebrate cat.

Although the gain of force feedback may very well be higher in other preparations, as discussed in SIMPLIFIED ANIMAL MODELS, p. 282, it would appear that the apparent regulation of stiffness observed in functionally isolated muscles is attributable by default to transmission through the spindle receptor pathway. Here it is important to note that linear feedback from spindle receptors cannot explain the compensatory effects. The component  $Ags$  in Equation 27 is a constant in a linear system and therefore cannot compensate for variations in  $K$ . This implication that nonlinear feedback from spindle receptors is an important mechanism in stiffness regulation is taken up again in Compensation for Yielding, p. 302.

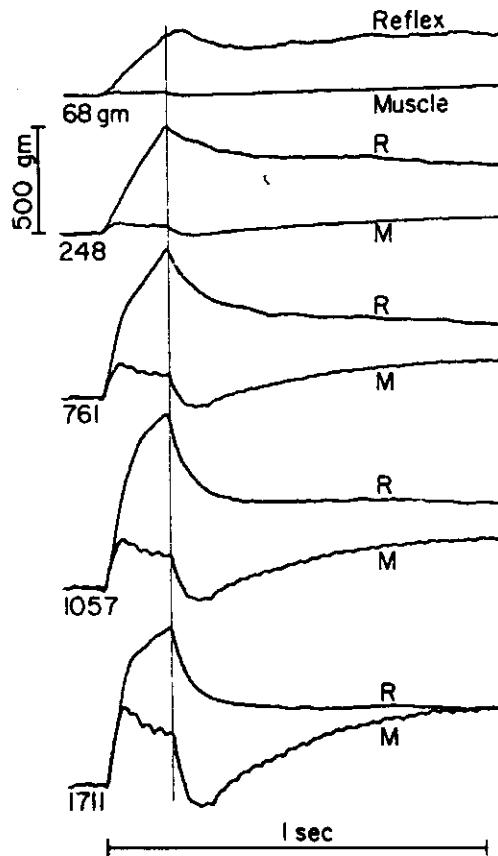


FIG. 27. Comparison of reflex and muscle mechanical responses at different levels of initial force. The input was a 2-mm ramp stretch applied at 12.5 mm/s to the soleus muscle. Initial force was modulated with a crossed-extension reflex to 5 different steady values in the case of the reflex responses. The muscle mechanical responses represent the force change recorded in an areflexive muscle that was electrically stimulated at 8 pulses/s. The mechanical response obtained was scaled (simulated recruitment) to match the initial conditions. [From Nichols (225).]

### Summary

The static properties of the motor servo are characterized by force-length curves that resemble muscle length-tension curves but are actually produced by a nonlinear pattern of motor unit recruitment. Many motor units are recruited as the muscle is first stretched past the threshold of the reflex, and most of the stiffness of the response in this region can be attributed to the neurally mediated component. At longer lengths, however, the mechanical stiffness of motor units that have already been recruited becomes progressively larger and tends to dominate the response at high force levels. The overall reflex stiffness is modestly dependent on initial force and relatively independent of muscle length. The fact that these appreciable dependencies are modest in comparison with the dependencies of muscle mechanical stiffness on initial force and length described in MUSCLE MECHANICAL STIFFNESS, p. 270, supports the hypothesis that autogenetic reflexes act to maintain the stiffness of the motor servo (*Stiffness Regulation*, p. 266). Since the loop gain of force feedback is small in functionally isolated muscles, the compensatory action must be attributed to nonlinear mechanisms in the spindle receptor pathway. The fact that the overall reflex stiffness is similar to and sometimes less than the stiffness of a maximally activated muscle is evidence against the length regulation hypothesis. If length were regulated, autogenetic reflexes should appreciably increase stiffness.

Experimental alterations in the control signals to the motor servo usually shift the threshold of the tonic

stretch reflex without appreciably altering the slope (stiffness) of the force-length relation. In terms of the nomenclature introduced in *Adaptive Models*, p. 269, these effects resemble the actions of motor commands rather than adaptive controls, in support of the summary model described on p. 268.

### STATIC REGULATORY CHARACTERISTICS IN INTACT SUBJECTS

Consideration of motor servo operations in intact subjects raises several new issues that were conveniently avoided in the previous section on functionally isolated muscles. These issues derive from the fact that muscles and their autogenetic regulatory loops are embedded within a complex motor system that engages in many control functions in addition to the local regulation of the length and force of individual muscles. Figure 28 summarizes some of the more basic features of added complexity, in this case arising from the mechanical and neural interactions between muscles that function as synergists or antagonists.

The mechanical linkage of the skeleton provides a mechanism for direct two-way interaction between muscles A and B (considered to be either synergists or antagonists), since both muscles apply forces on a common load and movement of the load affects the lengths of both muscles. Neural interaction arises from the projections of muscle proprioceptors onto synergistic and antagonistic motoneurons; these projections form closed feedback loops as a consequence of the mechanical linkages just mentioned. Since the signs of

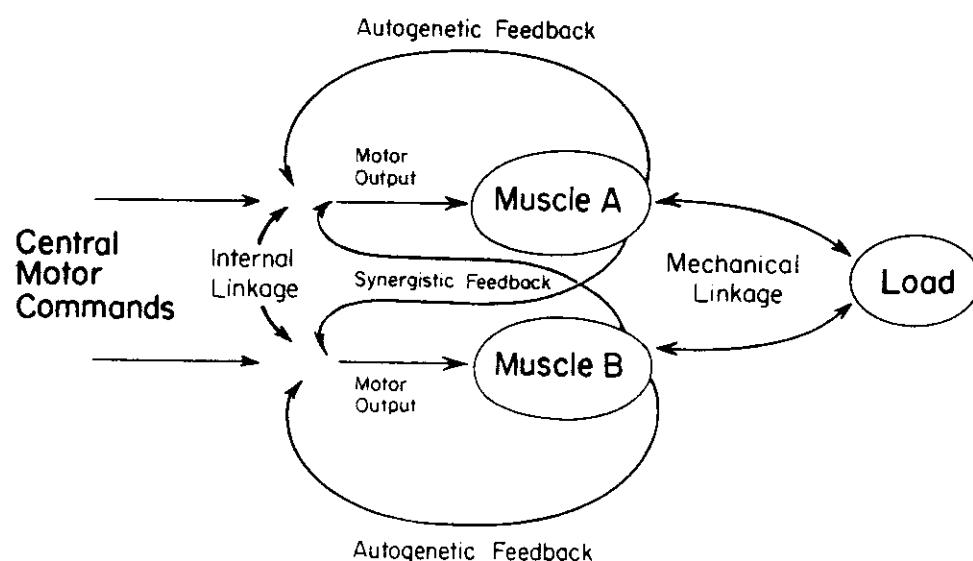


FIG. 28. Summary of relation between motor servos regulating contraction of single muscles (muscle A and muscle B) and their combined action as synergists. A given motor servo is represented by the loop that includes the muscle and its autogenetic feedback pathway. Factors that couple output from the two muscles are 1) both muscles act on the same load, 2) muscle afferent signals from one muscle are also distributed to the other muscle as synergistic feedback, 3) connections between neural elements of each motor servo (internal linkage) may coordinate action between muscles, and 4) central motor commands may be distributed to each motor servo.

the projections are the same as the signs of autogenetic projections for synergists and are in general opposite for antagonists [(58); see the chapter by Baldissara, Hultborn, and Illert in this *Handbook*], one would expect that the major effects would be to bolster the overall gain of length and force feedback as well as to facilitate spatial coordination among synergists, but these postulated functions have never been adequately evaluated. The internal linkages, such as the ones provided by Renshaw and Ia-interneurons (Baldissara, Hultborn, and Illert's chapter), provide additional channels for the coordination of muscle action about a joint.

#### *Skeletal Mechanics and Coordinate Systems*

Muscles insert at different distances from the center of rotation of a joint and the effective lever arm also varies with joint angle. Consequently, the forces developed by individual muscles cannot simply be summed to obtain a resultant force representing the combined actions of several agonists and antagonists. Summation is valid, however, if the forces are converted to torques (also called mechanical moments), which are the products of force times the effective lever arm for each muscle. A torque can be thought of as a force that promotes rotation, rather than linear motion, and the resultant torque is the sum of the individual contributions of agonists minus those of antagonists. The corresponding measure of rotational position is the angle of the joint, and the value that this angle assumes determines the lengths of each of the muscles via trigonometric equations. The fact that there can be only one joint angle guarantees that the lengths of muscles acting about a joint cannot be controlled individually, but only as a group.

Rotational movements about a single joint are relatively easy to analyze, but they are somewhat unnatural. Even the simplest movements generally involve several joints in some synergistic pattern, and there is evidence to suggest that the brain may control these multijoint synergies as facilely as it controls the synergies about a single joint (19). Hence, some investigators have chosen to study motor servo actions and movement-reflex interactions associated with multijoint synergies, and, as is shown by the examples in this section, the results appear to be quite compatible with ones obtained from experiments on single joints. Examples are the studies of head rotation by Bizzi, Polit, and Morasso (26) and of pushing and pulling movements of the arm by Evarts and Tanji (70) and by Gago, Houk, and Hasan (48).

#### *Steady-State Responses to Changes in Load Force*

Most investigators of functionally isolated muscles have studied force responses to imposed changes in muscle length as described in **TONIC STRETCH REFLEX IN FUNCTIONALLY ISOLATED MUSCLES**, p. 283, but in-

vestigators of stretch reflexes in intact subjects have more often chosen to study positional responses to changes in load force. Essentially the same information can be obtained with either approach, although the analysis of responses to force change is somewhat more complex due to the involvement of a mechanical load. As discussed earlier (*Summary Model*, p. 268), a mechanical load typically consists of both load properties and a load force (Figs. 1 and 12). In the simplest case the load properties are the inertia of the limb (or limb plus apparatus) and the load force is the result of gravity plus the force applied by the apparatus. The input consists of a change in load force, which, when applied to the inertial property of the load, causes an acceleration that progressively stretches the muscles and elicits a stretch reflex. A nontrivial problem addressed later (*Effect of Instructional Set*, p. 294) is that of insuring that subjects do not complicate their stretch reflexes by producing reaction-time movements.

Figure 29 shows the results obtained from the elbow musculature of two subjects using the protocol just outlined. The dashed step functions show the input changes in load force (positive forces tend to extend the arm) and the solid traces show, respectively, the arm forces resisting elbow extension, arm displacements, and the EMG responses of the biceps. Increases in load force accelerated the inertial load away from the subject, which elicited a stretch reflex in biceps. Decreases in load force permitted an excess of force of the elbow muscles to accelerate the inertia toward the body, which elicited an unloading reflex in biceps. In both cases the movement continued until arm force settled to a value appropriate to counterbalance the new load force, and this defined a new equilibrium state. The behavior of the arm as a whole was basically springlike, since a larger load force displaced it away from the body, whereas a smaller force permitted the arm to recoil toward the body.

The connection between these responses and the force-length relationship of the motor servo is elaborated in Figure 30, which is a simple adaptation of Figure 11. Imagine that the whole arm is replaced by a single muscle and that the solid force-length curve in Figure 30 represents the tonic stretch reflex of this muscle. The curve describes the reflex force that would be produced at any given length, whereas the force actually developed depends on an interaction with the mechanical load. At the initial load force designated, the muscle will be stretched, or will shorten to, the length indicated by the filled point, since only at this length will the reflex force be equal and opposite to the initial load force, insuring an equilibrium state. The responses to increases in load force shown in Figure 29 are approximately represented by trajectory a in Figure 30, which shows a transition to a new equilibrium. The muscle is stretched to a longer length at which it develops a larger reflex force, appropriate to counterbalance the larger force of the final load.

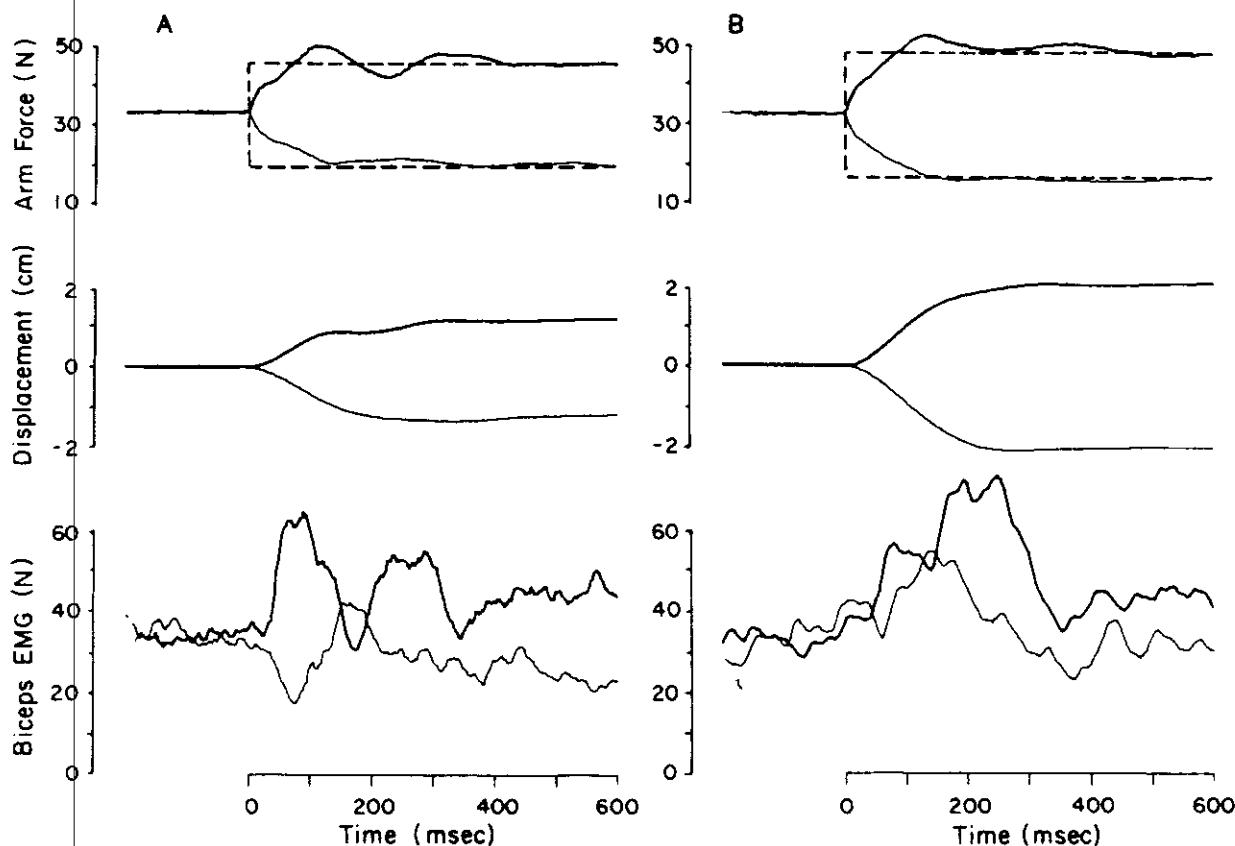


FIG. 29. Stretch and unloading reflexes in human arm. Symmetrical step increases or decreases in load force produce essentially symmetrical displacements of forearm, whereas biceps EMG responses are quite asymmetrical under these conditions. Step change in load force is depicted in *uppermost traces* as *dashed lines*, while resultant arm force, recorded by a load cell mounted within the apparatus, is a *continuous line*. *Heavy traces* represent responses to step increases in load force and *light traces* to equal but opposite decreases. EMG responses are full wave rectified and filtered (30 ms time constant) and calibrated in units of isometric force. Responses are ensemble averages taken from 2 different subjects using the no-intervention instruction (cf. Fig. 33). [From Crago, Houk, and Hasan (48).]

This example, though simplified, serves well to illustrate the interplay between the musculature and its mechanical load that determines the steady-state response to a change in load force. As such it provides a useful introduction to analogous interactions that have been characterized with torque-angle plots as described in the following section.

#### Torque-Angle Relations

Given that each of the motor servos regulating individual muscles acting about a joint is characterized by a force-length relation, the result of the combined regulations should be a torque-angle relation that serves to characterize the musculature of the joint as a whole (9, 72). The three solid curves in Figure 31 show a family of these torque-angle relations for the human elbow joint, analogous to the families of force-length curves described earlier. The positive slopes of the curves in both cases document a springlike restoring force. The particular joint angles at which torque

is zero are analogous to the threshold lengths in Figure 25, and the modulating influence of central motor commands in both cases causes, to a first approximation, a simple shift of the curve along the abscissa. The negative torques are a new feature that results from the activity of antagonist muscles.

These curves were obtained by first having the subject establish one of three initial postures defined by the points  $\alpha_6^{(1)}$ ,  $\alpha_6^{(2)}$ , or  $\alpha_6^{(3)}$ ; in other words the subject had to flex the elbow against a load in order to achieve one of three joint angles corresponding to one of three different levels of central motor command. Then the load was abruptly reduced to one of several lower levels, which allowed the elbow to flex further (biceps unloaded). The subject was told not to intervene voluntarily in order to suppress reaction-time (secondary) movements (cf. *Effect of Instructional Set*, p. 294). Each of the open circles shows a new value of torque and angle to which the arm settled after being unloaded, and the solid curves through these points define the torque-angle relations that

characterize the static properties of the elbow musculature under reflex control.

External loads can be characterized in terms of the static force or torque required to resist the load at each muscle length or position of the joint. For example, the line segments labeled a, b, and c in Figure 11 characterize the relation between force and length (Fig. 17) for an isometric load, a linear spring load, and a constant force load, respectively. Similarly the dashed curves in Figure 31 are a family of load lines that describe the elbow torque required to support different weights that were connected to the arm by way of pulleys. The required torque peaked at about  $120^\circ$  because the connecting cables were perpendicular to the arm, creating the greatest turning moment at this angle. Since the dashed curves represent the torque required at each angle and the solid curves described previously represent the torque actually produced by the musculature at each angle, a torque that is both required and available occurs only at joint angles corresponding to intersections of the relevant dashed and solid curves. Thus if the central motor command is at level (2) and the load is at level 3, the joint will settle to an angle of  $110^\circ$ , at which point the required and developed torque will both be  $0.4 \text{ kg} \cdot \text{m}$ . This point of equilibrium, marked  $\alpha_3^{(2)}$  in Figure 31, is stable, because at larger elbow angles the solid curve lies above the dashed curve insuring that the available torque will exceed the requirements for supporting the weight. Consequently the elbow will flex until the equilibrium between available and required torque is achieved.

Any change in load that occurs after the establish-

ment of an initial equilibrium will cause a transition to a new equilibrium. Thus if the load in the previous example were increased to level 4, there would be a transition from  $\alpha_3^{(2)}$  to the new equilibrium point  $\alpha_4^{(2)}$ . It is apparent from Figure 31 that this involves an extension of the elbow (stretch of the biceps muscle) and an increase in torque (due to a stretch reflex in biceps); the actual time course of motion would be like that shown by the stretch responses in Figure 29. In contrast, a reduction in load to level 2 would cause a transition from  $\alpha_3^{(2)}$  to the new equilibrium point  $\alpha_2^{(2)}$ . In this case the elbow flexes (biceps shortens) and the torque decreases (due to an unloading reflex in biceps), which is similar to the unloading responses in Figure 29.

#### *Equivalent Stiffness and the Concept of Composite Motor Servos*

The former results indicate that a springlike resistance to stretch and release is a consistent property of neuromuscular systems ranging in complexity from single functionally isolated muscles (Figs. 22-25) to sets of several synergistic and antagonistic muscles operating to control joint rotation (Fig. 31) or even whole-arm movements (Fig. 29). In analogy with the stiffness of single-muscle systems, the rotational stiffness of the joint (change in torque divided by change in angle) or the overall stiffness of the arm (change in the pulling force of the arm divided by the change in distance between the shoulder joint and the hand) are appropriate and useful summary measures of static regulatory performance. These equivalent stiffnesses can be conceptualized as the result of replacing all of the active muscles by springs, representing individual motor servos. Correspondingly the overall system of muscles and proprioceptive feedback pathways can be thought of as a composite motor servo having qualitative features quite analogous to a functionally isolated muscle, though perhaps showing quantitative differences.

The data required for a complete synthesis of a composite motor servo in terms of the properties of its constituent motor servos is not presently available. Nevertheless, Fel'dman (72) has shown that the observed curvature of the torque-angle relations is well accounted for by the curvature of force-length relations seen in functionally isolated muscles when these curves are combined with the geometry of muscle attachment to the skeleton. Another type of comparison can be made by converting equivalent stiffness to a normalized value. The stiffness of the whole arm reported by Crago, Houk, and Hasan (48) was about  $10 \text{ N/cm}$ , which when multiplied by the length range ( $40 \text{ cm}$ ) and divided by the force range ( $500 \text{ N}$ ) yields a normalized stiffness of 0.9, which is within the range noted earlier for functionally isolated muscles in decerebrate cats. Although this apparent constancy of normalized stiffness may be fortuitous, it does seem

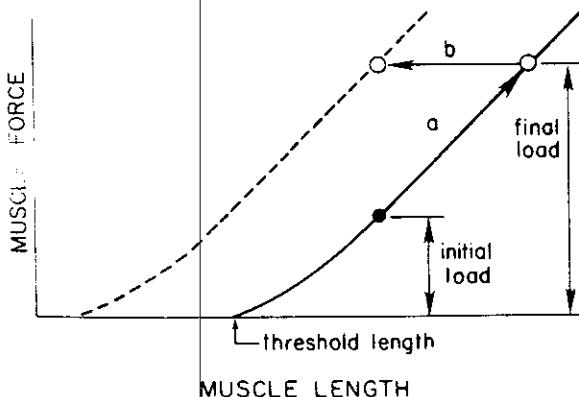


FIG. 30. Reflex (a) and reaction-time (b) components of response to a disturbance in force. Muscle length is determined by the intersection between the motor servo and the opposing load. ●, Initial equilibrium point, at which the force generated by the stretch reflex is equal and opposite to the initial load force. An increase in load force stretches the muscle along the force-length trajectory a, reaching a new equilibrium position (○), which is the point where final load is equal and opposite to stretch reflex force. The new equilibrium has been achieved at the expense of a significant length increase. The final load can be supported at the same length if the force-length relation is shifted to reach the position shown by dashed line. This shift may appear as a reaction-time movement causing shortening along trajectory b.

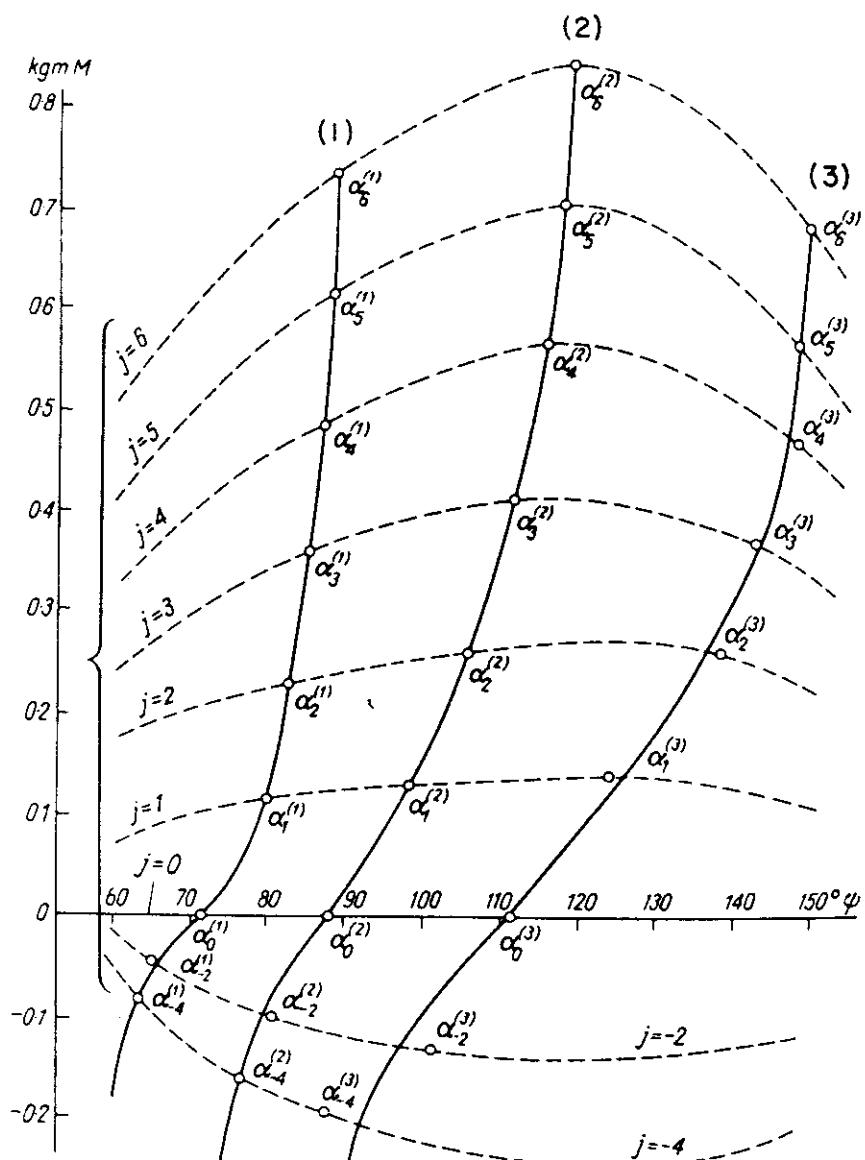


FIG. 31. Torque-angle curves relating the total moment developed by muscles at the elbow joint to the static joint angle, recorded following a series of load changes.  $\circ$ , Joint positions reached following the change in load. Solid lines connect the angles achieved following load changes introduced from particular initial angles  $\alpha_6^{(1)}$ ,  $\alpha_6^{(2)}$ , and  $\alpha_6^{(3)}$ . Dashed lines describe the external moments produced by the corresponding loads. [Adapted from Asatryan and Fel'dman (9).]

appropriate that muscle systems specialized to exert large forces also are endowed with a correspondingly large stiffness.

#### *Effect of Instructional Set*

The particular instructions given to the subject have an important influence on the responses obtained when a mechanical disturbance is applied to the limb or joint. An example of these effects of instructional set is shown in Figure 32, which is taken from a study of thumb responses to step increases in load force (191). In each of the three cases the initial effect of

the change in load force was the same—an extension of the thumb and a medium-latency EMG response in the stretched flexor pollicis longus muscle. At latencies longer than 117 ms the responses differed considerably depending on the prior instructions given to the subject. The latency at which instruction-dependent differences in EMG responses occur can be longer than this and can also be as short as 50–60 ms (114, 128).

One obvious source of these instructional effects is the subject's "willful" reactions to the disturbance (variously called reaction-time movements, voluntary responses, intended responses, or triggered responses), and another potential source is a preset change in the

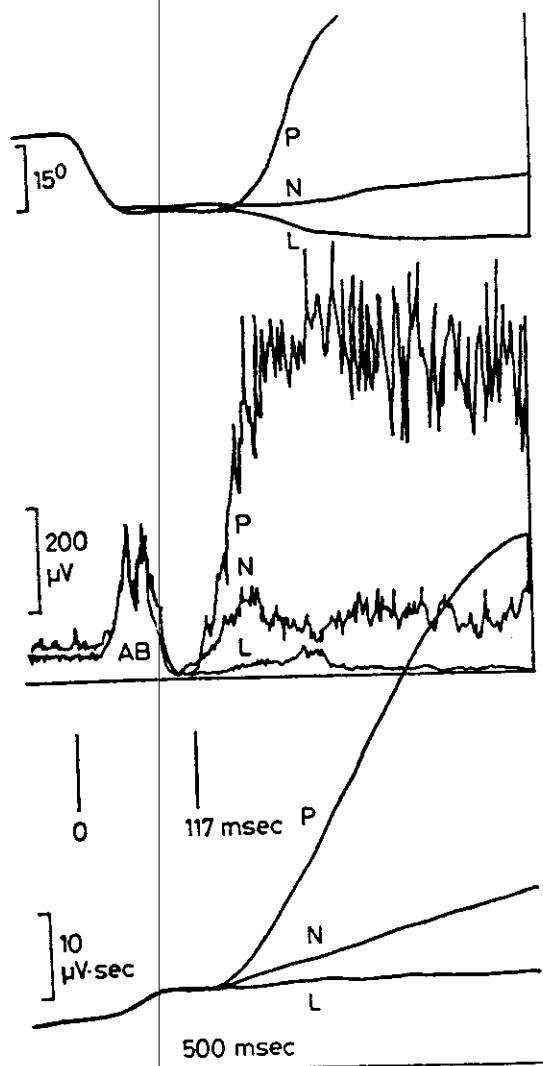


FIG. 32. The response to stretch of the long thumb flexor in human subjects depends upon prior instruction. At the outset, thumb is held flexed at  $151^\circ$ , supporting a force of 2 N, and it is then stretched abruptly at the point marked 0. Subject is instructed to either hold the thumb in a steady position (N), to relax (L), or to pull as hard as possible (P). The upper records show superimposed traces of angular position, the center traces show full wave rectified EMG (recorded from surface electrodes placed over flexor pollicis longus), and the lower traces are fully integrated EMG records. [From Marsden et al. (1971).]

sensitivity, or gain, of transmission through motor servo pathways. As discussed in *Adaptive Models*, p. 269, either or both mechanisms might contribute to the substantial adaptive capabilities of the motor system. It is important to distinguish between the two, since the underlying neural mechanisms are likely to be quite different. Furthermore there is the practical problem mentioned earlier that investigators studying the motor servo in intact subjects must be able to identify responses uncontaminated by reaction-time movements.

Hammond (114), who was one of the first to study

these problems, reported no effects of instructional set on the short-latency responses of the human biceps muscle, but rather substantial effects on medium-latency and later components (using the terminology described in *Long-Loop Reflexes*, p. 280). Medium- and long-latency components were large when the subjects had been told to resist the disturbances and were sometimes completely inhibited when the subjects had been told to let go. Similar findings have been reported from several laboratories (48, 67-71, 93, 168, 191), although it should be noted that the instructional effects sometimes occur only in the long-latency components (as in Fig. 32). Some authors have reported that the short-latency component is also modulated by instructional set (67-70, 168), but these effects do not appear when the initial force of the limb is controlled. Variations in response associated with different initial forces probably result from system nonlinearities rather than from adaptive control mechanisms, as discussed in the following section.

The dependence of the medium-latency response on instructional set was originally interpreted as evidence for an adaptive gain-control mechanism (114). This conclusion was based on the assumption that reaction times are too slow to influence the amplitudes of medium-latency responses. By default the instructional effects on these components were attributed to adaptive changes in the gain of a length-feedback loop, and it was further postulated that gain control might be mediated by a transcortical servo loop (67, 236).

Distinguishing between a gain-control mechanism and a reaction-time process based on latency has two major disadvantages. One is that it relies heavily on a valid measure of a minimal reaction time and the other is that this approach is incapable of partitioning later components of response. Reaction times vary considerably with the modality and intensity of the stimulus and with several other parameters (see the chapters by Poulton and by Keele in this *Handbook*). The usual procedure in stretch-reflex studies has been to measure a reaction time to stimuli other than muscle stretch or to small tendon taps that provoke no appreciable reflex and to assume that the latency of a reaction in response to a large stretch would be no shorter (e.g. refs 114, 187, 206). Recent experiments in which alternative criteria were used to identify reaction-time mechanisms have demonstrated shorter latencies (48, 68), ones that in some cases are sufficiently fast to modulate the amplitude of medium-latency EMG responses (128). One of these alternative approaches is particularly well suited to partitioning the overall response into motor servo and reaction-time components, as illustrated in Figure 33.

This figure shows, for each of two subjects, six superimposed arm position responses to a standard step increase in load force, obtained with two different instructional sets. Simple, consistent, springlike responses were obtained with the no-intervention instruction, whereas more complex and more variable

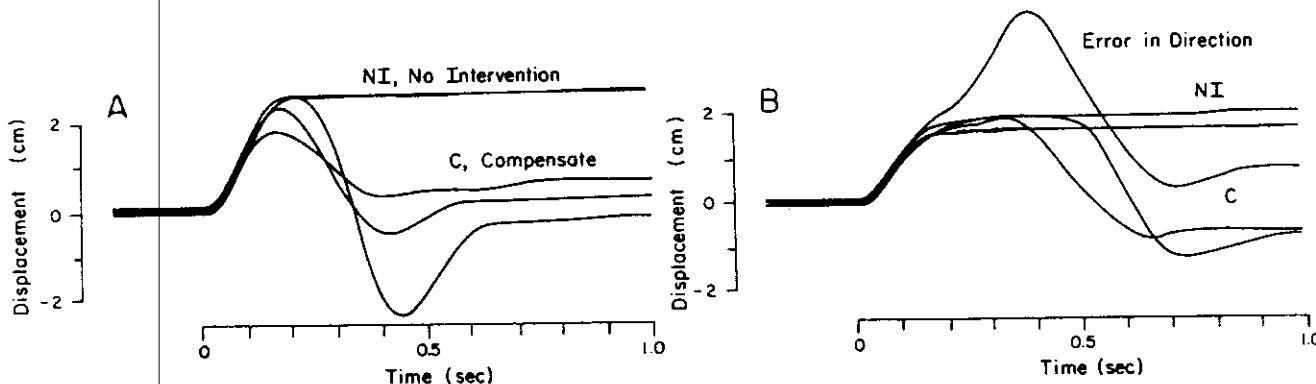


FIG. 33. Features of reaction-time movements of the arm, initiated in response to step change in load force. Force change occurs at time zero. The subject is told either to compensate (C) or "do not intervene voluntarily" (NI). In the latter case, both subjects (A, B) produced simple springlike response (NI traces). When the subject attempts to compensate for the perturbation (C), the initial trajectory is similar to that of NI but deviates at a point whose latency varies from trial to trial and with increasing choice (compensate vs. do not intervene). Upper trace in B depicts an inappropriate response, obtained with the compensate instruction. Records are of single trials, following  $\pm 18$  N load changes in A and  $\pm 15$  N change in B. [From Crago, Houk, and Hasan (48).]

responses were obtained with instructions to compensate or resist. The latter responses depart from the common initial phases of arm deflection at points that have been identified as the onsets of reaction-time movements based on three criteria. One is the variability of this latency, which was deliberately accentuated in these trials by introducing stimulus uncertainty. Subjects did not know whether the impending disturbance would deflect their arms away from the body (as in the trials shown) or toward the body (not illustrated). A second criterion is a shortening of the latency of the instructional effect that can occur when stimulus uncertainty is eliminated by using a single direction of disturbance. A third criterion is the presence of occasional errors in the direction of the compensatory response (Fig. 33, right panel). Stimulus uncertainty creates a choice situation that can have marked effects on the performance of a reaction-time process, whereas one would not expect any change in the operation of a servo loop in which the gain has been preset to increase the rigidity of the arm. Thus the increased latency, the variability, and the errors in direction observed are all indicative of a reaction-time mechanism. The absence of any fixed-latency instructional effect argues against the hypothesis for gain control. It would appear that the most recent experiments by the Marsden group (255) also favor this conclusion.

The instruction "do not intervene voluntarily" was first introduced by Asatryan and Fel'dman in 1965 (9) as a method for getting subjects to refrain from producing reaction-time movements, leaving relatively pure motor servo responses. The variability comparisons discussed in the previous paragraph lend support to the validity of this procedure and hence to the validity of the assumption that the torque-angle relations discussed on p. 292 represent motor servo prop-

erties. Nevertheless, some subjects are reported to have considerable difficulty in suppressing the superimposed reaction-time components, which suggests considerable automaticity in the underlying neural process (48, 128). The thumb-position responses obtained with the instruction "hold firm" (trace N in Fig. 32) appear to represent relatively pure motor servo responses; they are simple, springlike deflections that appear to be analogous to the no-intervention responses in Figure 33. Without evidence to the contrary, it seems reasonable to accept low variability and springlike properties as being indicative of motor servo responses in intact subjects.

#### *Gain Variation vs. Gain Control*

The results reviewed in the previous section suggest that effects of instructional set previously thought to demonstrate gain control instead may be attributable to short-latency reaction-time movements. Although these data do not exclude gain change as a mode of adaptive control, this possibility becomes less likely. Here we review other results in which there are apparent changes in gain, and we discuss whether or not the observations constitute evidence for gain control.

Several authors have demonstrated that the short- and medium-latency EMG responses to a standard disturbance undergo regular variation as the initial force is altered (91-95, 185, 186). Furthermore it is apparent from Figure 31 that the rotational stiffness of the elbow (slope of torque-angle curve) varies with initial torque in much the same way as the static incremental stiffness of a functionally isolated muscle varies with initial force (*Dependence of Incremental Stiffness on Initial Force*, p. 287). These variations are due at least in part to two major nonlinearities, the proportionality between the mechanical compo-

ment and initial force (*Natural Combinations of Recruitment and Rate Modulation*, p. 276) and the recruitment nonlinearity (*Mechanically and Neurally Mediated Components*, p. 285). Gain variations associated with built-in nonlinear properties such as these cannot be controlled in any independent fashion and therefore do not constitute mechanisms for gain control. As discussed in *Adaptive Models*, p. 269, the adaptive control of gain requires a mechanism for adjusting gain independently of the central motor command that controls stretch reflex threshold. It follows that one should be able to demonstrate changes in stiffness while holding the initial mechanical conditions constant.

Changes in stiffness have been reported to occur without any change in the net initial torque or force of the limb in association with cocontraction of agonists and antagonists. Asatryan and Fel'dman (9) found that cocontraction increased the slopes of torque-angle curves in the vicinity of the zero net torque points, whereas the curves were not appreciably altered for torque values above about 10% of maximum. These changes can be accounted for by assuming that cocontraction is produced by adjusting the reflex thresholds of both agonists and antagonists to shorter muscle lengths, without altering the stiffness relations for either muscle group (72). Crago, Houk, and Hasan (48) reported that most subjects were not very effective at contracting when the arm was initially loaded with 10% maximal force. For those who succeeded, the stiffness of the arm was approximately doubled, apparently as a mechanical consequence of cocontraction, for the EMG changes observed during test stretch reflexes were not appreciably altered. These various results indicate that a limited degree of adaptive control over limb stiffness can be achieved by the mechanism of cocontraction. This mechanism is particularly important at low levels of initial force (i.e., in initially unloaded limbs).

There are many reports in the literature describing apparent changes in gain when stretch reflexes are elicited in initially inactive limbs. While it is true that the initial force is kept constant at zero in these cases, the inactive state fails to control the initial bias of the motoneuron pool. An identical afferent input can be expected to produce a larger response if the motor pool is biased near its threshold than if the pool is biased well below threshold, as would be the case in a totally relaxed state. Such effects may be important in preparing a subject for a transition from rest to activity, but they do not demonstrate an independent mechanism for gain control.

It is possible that gain-control mechanisms are activated subconsciously during appropriate postural tasks but are inaccessible to instructional set. Nashner (221) has described marked adaptive changes in the reflex responses to ankle rotation in free-standing subjects, but it is not certain whether these responses

represent motor servo actions or triggered reactions (cf. ref. 94). The fact that the EMG activity occurs in fixed patterns of output to whole groups of leg muscles (222) is suggestive evidence for the involvement of a motor program, rather than a servoregulatory action.

### Summary

The results reviewed in **TONIC STRETCH REFLEX IN FUNCTIONALLY ISOLATED MUSCLES**, p. 283, and in the present section suggest a great deal of similarity between the static properties of the motor servo in functionally isolated muscles and in muscle systems of intact subjects. In both cases the behavior is basically springlike, and control signals act mainly by altering the slack length of the equivalent spring, rather than by modifying the stiffness of the system. The major exception to this rule concerns the use of cocontraction to stiffen unloaded limbs in preparation for disturbances. Variations in motor servo gain occur in association with system nonlinearities, but there seem to be no clear examples to document gain control, which, in theory, could be an effective mechanism for modifying the rigidity of the limb. Although this negative result may simply reflect a failure to investigate appropriate situations, it does seem clear that this mode of adaptive control is not as prevalent as was once believed.

### DYNAMIC RESPONSES TO MECHANICAL DISTURBANCES

In this section we describe the dynamic responses of the motor servo to imposed changes in muscle length and to changes in load, and we attempt to relate some of the characteristic features of these responses to specific afferent and efferent mechanisms. We show that some features derive from the response properties of muscle proprioceptors, whereas others relate more to muscle mechanical properties.

#### Dynamic Features of Force Development

As emphasized earlier, the increment in force produced by a muscle during a stretch reflex consists of a mechanical component, mediated by the mechanical properties of motor units that are initially active, and a reflex-action component, mediated by the recruitment of additional motor units and by increases in the discharge rates of units already recruited (cf. Eq. 13, and *Mechanically and Neurally Mediated Components*, p. 285). Recruitment and rate modulation represent the efferent mechanisms for the implementation of motor servo error signals. While we have discussed the mechanical responses to stretch and release in some detail (**MUSCLE MECHANICAL STIFFNESS**, p. 270), we have not yet given much attention to the effects of muscle mechanical properties on the translation of motor servo error signals into reflex action.

In many respects, muscle may be treated as a low-pass filter, which is to say that rapid fluctuations in the stimulus rate are smoothed and attenuated in the force output (see the chapter by Partridge and Benton in this *Handbook*). A clear example of this low-pass property is illustrated in Figure 13 in which the onset of a tetanic stimulus to the muscle nerve (which amounts to a step change in muscle excitation) induces a gradual and progressive increase in isometric muscle force. The time course of this smoothing effect is quite prolonged, since the final steady force may not be achieved until several hundred milliseconds after stimulus onset (depending upon the stimulus frequency). A comparable prolonged decline in force also follows stimulus cessation. These types of force prolongation would evidently smooth out fluctuations in motor output, but they must also limit the speed with which a motor servo correction can be executed.

In most published studies the low-pass properties of muscle have been quantified with frequency-response techniques, in which stimulus trains modulated over a wide range of sinusoidal frequencies are applied to motor axons, and the resultant force fluctuations are measured. To summarize some of the major results, when a train of stimulus pulses is subjected to sinusoidal rate modulation around a mean rate, the force output of the isometric muscle is also sinusoidal in time course, but peak-to-peak force modulation (which is a measure of system gain) declines steeply above a corner frequency (16, 66, 231, 242, 254). Similar findings were reported by Mannard and Stein (184) using a stimulus train whose rate was modulated by a random noise signal. The corner frequency varied among different muscles but lay in the range of 2–7 Hz in all cases. The results of these various frequency-response studies strongly support the low-pass filter analogy and show that the relation between isometric force change and stimulus input is well fitted by a linear second-order model whose properties may be defined by a low-frequency gain, natural frequency, and damping parameters (184). For the plantaris nerve-muscle preparation, typical values were: low-frequency gain  $63 \text{ g} \cdot \text{impulse}^{-1} \cdot \text{s}$ , natural frequency 5.0 Hz, and damping ratio 1.0.

Although the predictions provided by the linear models of isometric contraction are often close to the recorded force measurements, significant deviations from linearity do arise, particularly in response to high-frequency stimulus components. For example, very closely spaced stimuli, called doublets, give rise to unpredicted and sustained increases in isometric force. This phenomenon has been termed the catch property (289), and it is found in both mammalian (36, 109) and invertebrate muscle systems (289). Furthermore the relation between tetanic force and stimulus rate is not well fitted by any second-order linear model (233); during tetanic stimulation the magnitude of the force increase predicted by available models is considerably greater than that recorded under experimental

conditions. In addition, the frequency-response function can also be shown to depend on the mean stimulus rate (184) and on absolute muscle length (248, 254), which is indicative of nonlinear behavior.

All of the latter studies deal with responses of isometric muscle, whereas, under most physiologically relevant conditions, changes in motoneuronal output arise in the course of ongoing variations in muscle length and tension. We have already described several important nonlinear features of the muscle response to lengthening; however, with some important exceptions [(232); cf. the chapter by Partridge and Benton in this *Handbook*], the response to joint variations in stimulus input and length has not been explored. Preliminary evidence indicates that one major nonlinearity, muscle yield, is minimized or even eliminated when muscle activation and stretch occur simultaneously (46, 47). In spite of the deficiencies of linear models (cited in previous paragraphs), a linear second-order transfer function for the stimulus-force relation remains a useful, if incomplete, description.

The low-pass features of the muscle response to neural input are not discernible in the reflex (242). When the functionally isolated muscle is subjected to sinusoidal length change, the rectified EMG shows a frequency-response curve in which gain rises rapidly above a corner frequency of 2 Hz. This EMG gain, which is analogous to that of a high-pass filter, counteracts the declining gain of muscle response to neural input at high frequency, so that the net gain remains essentially constant over much of the physiologically relevant frequency range. The high-pass feature of the EMG response appears to originate in the properties of muscle spindle receptors (242, 267).

#### Dependence of Transient Responses on Initial Force

In *Dependence of Incremental Stiffness on Initial Force*, p. 287, we reviewed the observation that the static incremental stiffness of the reflex response varies with the initial force level, rising at low initial forces, reaching an essentially stable value over much of the range of force modulation, and tapering off to somewhat lower values at near maximal forces (Fig. 26). It was pointed out that these variations are relatively minor in comparison with the approximately proportional dependence on initial force of the mechanical component. Figure 27 also displays broadly comparable changes in the dynamic incremental stiffness as the initial force is increased. When dynamic stiffness is measured at a constant point (ramp termination, for example), the stiffness is seen to increase with increasing initial force, being approximately 1.35 N/mm at the lowest initial force, then rising to a maximum of approximately 4 N/mm at an initial force of about 10.5 N. Even higher initial forces are associated with stable or falling stiffnesses, the latter resulting presumably from saturation of motor output mech-

isms. These stiffness calculations also reveal that, though the increase in stiffness is substantial, it is not in proportion with initial force as would have been anticipated if the response were purely mechanical (cf. *Natural Combinations of Recruitment and Rate Modulation*, p. 276).

This difference between force and stiffness could result from a variety of factors. For example, as initial force rises, an increasing contribution from rate modulation of active motor units is to be expected, because the motor pool available for recruitment is progressively depleted; initial force increases resulting from rate modulation would not necessarily be associated with transient responses that scale with initial force, as was the case with recruitment. This relation between force and stiffness is as likely to be caused by nonlinearities in recruitment of new motor units, as by force-feedback contributions. These alternatives are evaluated at a later point; however, it is first necessary to further dissect the mechanical and reflex contributions to motor servo output.

To return to Figure 27, if we compare the total dynamic force output with the predicted muscle mechanical response, it becomes apparent that the mechanical contribution provides a greater and greater fraction of total force output as initial force is increased. In fact, the mechanical response provides the dominant contribution at the highest initial force level (1.711 g), contributing more than half the dynamic force increase, and essentially all of the static force. The increasing mechanical contribution is also accompanied by a progressive reduction in the magnitude of reflex action, which is probably responsible for the lack of scaling of the whole force response with initial force level. These data do not allow us to distinguish between reductions in reflex output arising from force feedback and reductions arising as a result of nonlinearities in the control of motor output.

The figure also shows that there are significant deviations in the time course of the reflex response that accompany increasing initial force. For example, the steepness of the initial rise in force increases at the higher force levels, but this region of high stiffness is then interrupted by a transition to a region of much lower stiffness. This two-phase dynamic response is most distinct at the highest initial force, as the mechanical component becomes more and more dominant. There are also interesting changes in the time course of postramp adaptation. The magnitude of adaptation is negligible at the lower force levels, but it becomes quite prominent at the highest force levels, presumably as a consequence of the larger adaptation in underlying muscle mechanical response.

In sum, it is evident that the stiffness of motor servo output is not constant in the face of changing initial force levels. The changes in stiffness appear to be relatively modest, however, and there are substantial regions of response in which changes in initial force induce negligible changes in stiffness. At the present

time, the relative contributions of force feedback and output nonlinearities in this apparent stiffness regulation are unknown.

#### *Amplitude Dependence and Linearity*

In sections *Instantaneous Stiffness and Short-Range Elasticity*, p. 272, *Instantaneous Stiffness ... Region*, p. 274, and *Ramp Responses ... Nonlinearity*, p. 274, the stiffness of electrically stimulated muscle was shown to vary markedly with the amplitude, direction, and velocity of imposed length change. Comparison of these nonlinear properties with the dynamic response of muscle under reflex control illustrates that these dependencies are markedly reduced in the presence of reflex action. Figure 34 shows the response of normally innervated soleus to ramp stretches of varying amplitude, together with the predicted mechanical component. Stretch amplitudes varied from 0.2 mm to 3.4 mm, encompassing about 0.5%-10% of the maximum physiological range of movement, and were of fixed duration (160 ms). As in Figure 27, the predicted force output of that portion of the muscle that was active before the length change occurred (i.e., the muscle mechanical component) is superimposed on each reflex response. Estimates of the mechanical component were derived from measurements on electrically stimulated muscles, based on the assumptions that motor unit discharge rate was 8 pulses/s (107) and that increases in initial force resulted largely from activation of increasing fractions of the motoneuron pool. The accuracy of this approach to the prediction of muscle mechanical response is attested to by the superposition of the initial portions of the muscle and reflex responses.

A comparison of the reflex responses with the mechanical components reveals that the initial region of high stiffness in the reflex response extends smoothly beyond the point where yield would be expected to take place; no discontinuity is evident in the force records. This smooth force trajectory is not uniformly present in all responses, however. Under certain conditions such as very rapid stretch, very high initial force levels, or poorly functioning reflexes, discontinuities do appear on the force trace and these correspond quite closely in timing and magnitude with the predicted point of yield (226). These examples in which force discontinuities are detectable are useful because they provide strong evidence supporting the validity of the estimates of the underlying mechanical component of response.

In previous sections we have shown that motor servo action serves to minimize the dependence of responses to stretch on the initial force and length of muscle. Similarly, Figure 34 illustrates that in the presence of motor servo action the force change evoked during dynamic stretch or release is more closely proportional to the amplitude of length change than is the mechanical component. The relation be-

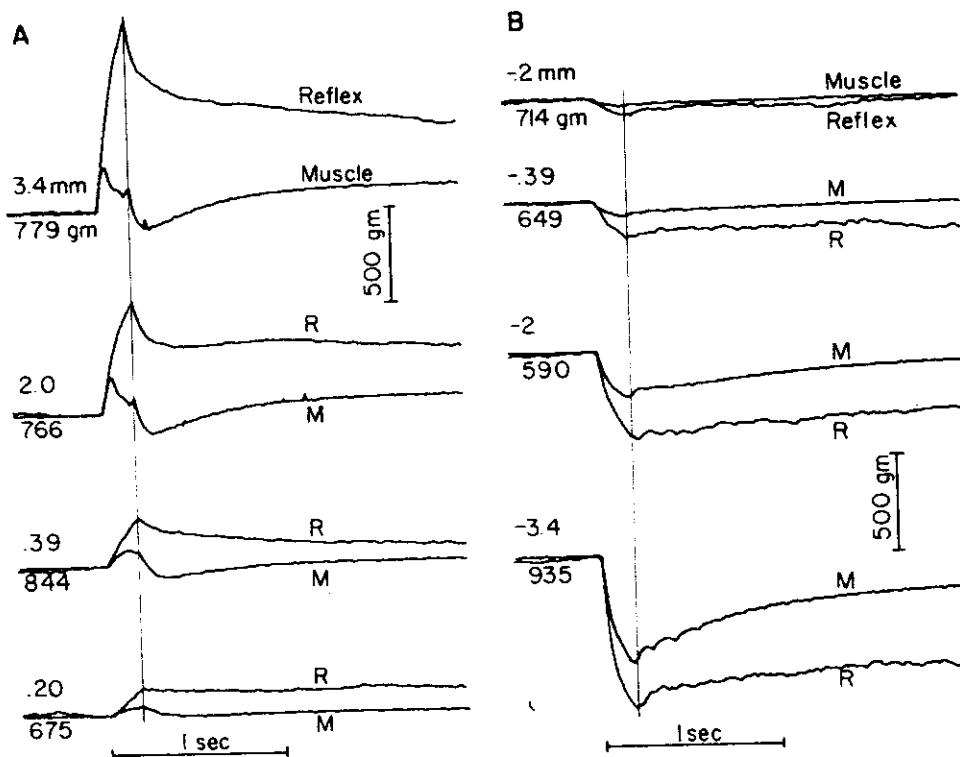


FIG. 34. Dependence of reflex (R) and mechanical (M) responses on the amplitude and direction of length change of soleus muscle in the decerebrate cat. Ramp changes in length had the same duration (160 ms) but were of differing amplitude. The amplitude of length change and the initial force are shown to the left of each record. The mechanical response (M) was derived from a different soleus muscle during stimulation of the ventral roots in a distributed manner at 8 pulses/s. Records in A represent responses to stretch, while those in B represent response to symmetrical release. [From Nichols and Houk (226).]

tween dynamic force and length becomes generally springlike, in that there is a roughly proportionate increase in force with increasing stretch amplitude. This relative constancy of stiffness is especially noteworthy during the dynamic phase of stretch when the stiffness of the mechanical response shows a marked reduction. A more precise assessment, however, provided by plots of force increment versus length increment (226) reveals that the stiffness is not quite constant, but is greatest for small amplitudes of stretch and release and then decreases moderately for increasing amplitudes of length change. Stiffness is also greatest during the dynamic phase of movement and then declines somewhat following ramp termination, particularly after large stretches (compare force response to 0.2 mm and 3.4 mm stretch in Fig. 34). However, these amplitude- and time-dependent variations in stiffness are minor when compared with the variations in stiffness of the underlying mechanical response discussed in *Ramp Responses: Transient Properties and Non-linearity*, p. 274.

An analysis of linearity similar to that described for muscle in *Ramp Responses ... Nonlinearity*, p. 274, has been used to demonstrate that the initial region of high dynamic stiffness extends for about 400–500  $\mu$ m

(226). This is about 1%–2% of the physiological range, a value commensurate with the amplitude of short-range stiffness observed in active areflexive muscle. If stretch amplitude exceeds this value, the stiffness shows a progressive 3- to 4-fold reduction. Similar amplitude dependence was first reported by Matthews (97), who showed that the soleus reflex stiffness associated with step changes in length remained high for up to 0.4 mm of stretch. While the high stiffness in the dynamic phase is probably attributable to muscle mechanics, high stiffness in the period following length change must be attributed largely to reflex action, because the static mechanical contribution is insignificant for these amplitudes of stretch. The enhanced sensitivity of primary endings to small amplitude stretch is undoubtedly a factor, although it is noteworthy that the region of high stiffness exceeds the small signal region of primary endings by a considerable margin (see below in this section). A secondary ending effect on spinal motor output may also be involved. In sum, while the initial transient region of high stiffness seems to be due to the mechanical properties of muscle, the augmentation that persists during small amplitude stretch is undoubtedly of reflex origin.

The linearity and amplitude dependence of the reflex response have also been studied with sinusoidal perturbations of muscle length or force in functionally isolated cat muscles (21, 242, 254) and in intact limbs of human and animal subjects [(20, 86); the chapter by Rack in this *Handbook*]. For example, Goodwin and colleagues (86) applied sinusoidal length perturbations to jaw-closing muscles in the primate and observed much larger stiffnesses for small movements (100  $\mu\text{m}$  peak to peak) than for large movements (500  $\mu\text{m}$ ) over a wide range of sinusoidal frequencies. These amplitude-dependent variations were shown to depend on motor servo action, because they were eliminated by electrolytic lesions of the muscle afferent projection pathways. Similar amplitude-dependent changes in stiffness of reflexively activated cat muscles were noted by Berthoz and co-workers (20) in the course of sinusoidal variations in load force. In contrast, in the study by Rosenthal and colleagues (254), also performed in hindlimb muscles of the decerebrate cat preparation, the stiffness of reflex response to sinusoidal length change did not show comparable amplitude dependence. The force variation remained linearly dependent on amplitude for peak-to-peak movements of almost 3 mm over a wide range of frequencies. The reason for this difference in amplitude dependence is not known.

Finally, it is of some interest that the linear response region of muscle spindle receptors is even smaller than that of contracting areflexive muscle. For spindle receptors, the linear region is defined as the amplitude of stretch within which discharge rate remains proportional to the amplitude of length change—in the case of soleus primary endings, this amplitude is approximately 100  $\mu\text{m}$  (116, 117, 202). It appears, therefore, that the motor servo is able to combine several nonlinear elements to provide an improved range of linear behavior. Some ways in which such cascaded nonlinearities might interact to increase the range of linear response is discussed in the next sections.

#### Asymmetry of Motor Servo Response

Although the force output in functionally isolated muscles recorded during stretch and release of equal amplitude is rather symmetrical, the EMG response assessed under the same conditions turns out to be quite asymmetrical (226). The existence of asymmetrical EMG response can be predicted from the fact that reflex action, which is equal to the difference between the mechanical component of response and the total reflex force, is much larger during stretch than it is during release (Fig. 35A). The EMG asymmetry should be opposite to that of the underlying muscle mechanical response to produce the observed asymmetry of reflex action. Similar results have been obtained in studies of the elbow musculature in humans (48). The EMG responses of biceps (Fig. 29) show greater increases during stretch, when muscle

yield should be prominent, than decreases during release, when muscle shows less drastic reductions in stiffness. In fact, part B of this illustration shows that in some subjects the EMG may even increase substantially during release. Leaving the issue of mechanism aside for the moment, it should be noted that this type of asymmetry is not appropriate for a length servomechanism. During externally imposed muscle shortening, a length servo might be expected to withdraw muscle excitation, since persistent or increasing muscle excitation would assist further muscle shortening, rather than the restoration of the muscle to its original length. The pattern of the asymmetry is much more appropriate for a mechanism regulating stiffness, in that the EMG change appears tailored to preserve the symmetry in the mechanical stiffness of muscle.

The physiological basis of this asymmetrical EMG response has been subjected to little direct study; however, several possible mechanisms warrant consideration. First, it is conceivable that asymmetry of the muscle mechanical response gives rise to differences in the force-feedback signal during stretch and release, which in turn might be responsible for asymmetrical features of the motor output. This mechanism, however, is probably not the source of asymmetry, because calculations based on the estimated loop gain of force feedback in the decerebrate cat predict less regulation than is actually observed (226). Nevertheless, it has been pointed out that the augmentation of EMG during release of the human arm (depicted in Fig. 29B) could be a consequence of reduced activity of tendon organs; and, if so, this would suggest that force feedback has a higher gain in intact subjects than in the decerebrate subject.

A second possibility is that the EMG asymmetry could result from asymmetrical spindle receptor responses to stretch and release of muscle. During stretch, the primary ending responds by producing a substantial dynamic response, while during release it will often cease firing. In other words, asymmetry could develop because the shortening spindle receptor might drop below threshold producing a lower bound saturation (or rectification). This saturation would be most prominent when initial discharge rate is low and when shortening velocities and shortening amplitudes are large. The introduction of substantial levels of  $\gamma$ -static and  $\gamma$ -dynamic activity should only exacerbate the asymmetry, because  $\gamma$ -dynamic activity selectively augments the primary ending response to stretch (139), whereas  $\gamma$ -static activity reduces the rate decrease during muscle shortening (139). The postulated asymmetrical features of primary ending responsiveness have been observed, as shown in Figure 35B. Comparison of the reflex responses in part A with the spindle receptor responses in B, makes the point that the asymmetry of reflex action and the asymmetry of primary endings are broadly comparable. The response of secondary endings under these conditions is much more symmetrical, even in the presence of con-

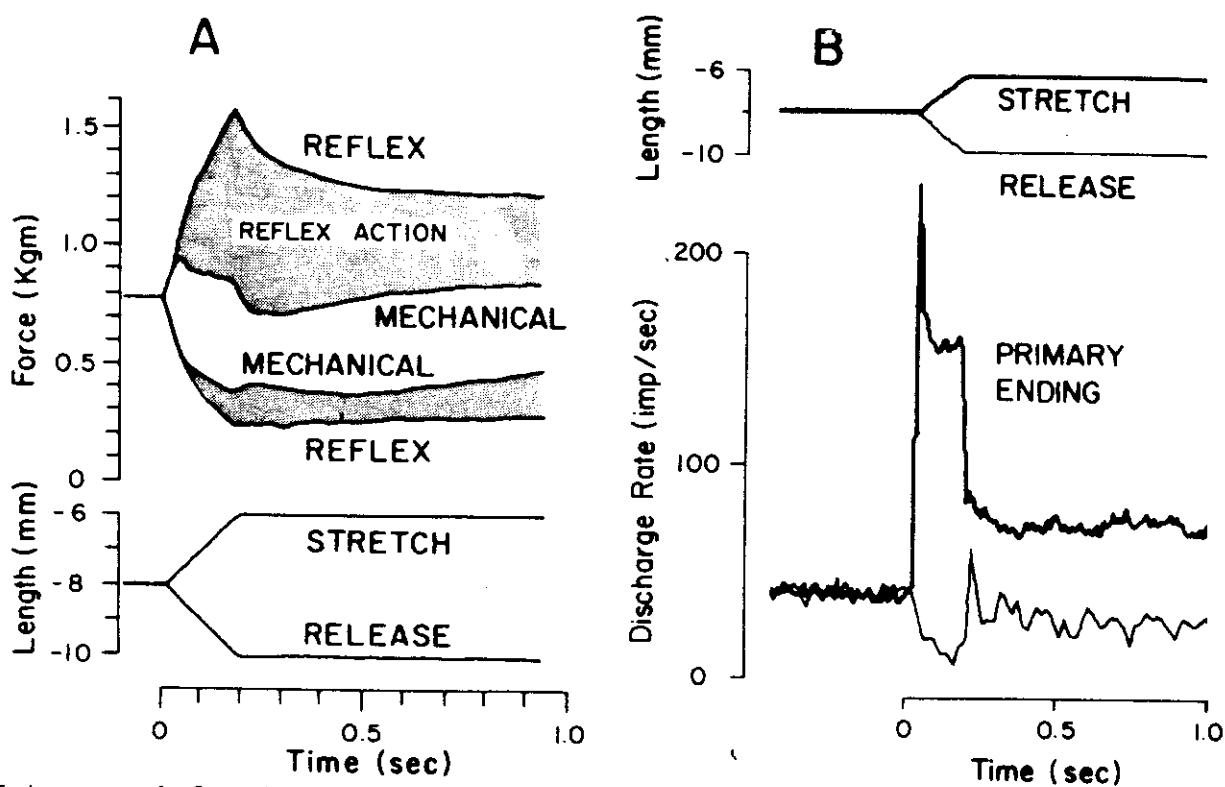


FIG. 35. Asymmetry of reflex action and its origin in primary endings. The response of soleus muscle in the decerebrate cat to stretch and release applied at 12.5 mm/s are compared in A. The responses labeled mechanical represent the changes in force that would occur if there were no reflexly generated changes in motor unit discharge or in the number of motor units recruited. Reflex action is the difference between the overall reflex and its mechanical component. Part B illustrates the response of primary spindle afferent, examined under closely comparable conditions. The greater reflex action associated with stretch, as contrasted with release, appears to be due mainly to the greater dynamic response of primary endings to stretch, as contrasted with release. [From Houk, Rymer, and Crago (137).]

siderable fusimotor drive (134), making them a less likely potential source of EMG asymmetry.

One should also consider the possibility that, due to nonlinearities or selective sensitivities, the tendon organ pathway operates in an asymmetric manner. Tendon organ discharge has been shown to provide an excellent sample of instantaneous total muscle force, with some dynamic sensitivity, during reflexively activated muscle contractions (133) and during locomotion (245). Tendon organ response to rising versus declining muscle force is also relatively symmetrical under both static and dynamic conditions, and there seems to be no evidence that the tendon organ is selectively responsive to the mechanical component of reflex responses (134). On present evidence, therefore, it seems unlikely that tendon organ receptors are an important source of EMG asymmetry.

While the asymmetry of the primary ending response to stretch and release is very likely to contribute to the asymmetry of the EMG, other mechanisms could also be important. For example, interneurons and motoneurons could also introduce a rectifying stage when they fall below threshold during phases of

declining excitation. In those circumstances where spinal excitability is sufficient to prevent such a lower bound saturation, existing evidence indicates that spinal transmission is relatively linear (242, 287).

#### Compensation for Yielding

In *Stiffness Regulation*, p. 266, we described one possible scheme in which regulation of this property might arise as the result of linear feedback from receptors sensitive to length and force. This arrangement, which follows from the layout in Figure 1, is that of a linear force-length comparator, in which the values of muscle length and/or force are sensed and compared before an error signal can be computed. There are features of the transient responses, however, that are not well accounted for by the linear force-length comparator notion. One is that the magnitude of the asymmetry in reflex action discussed in the previous section is too large to be accounted for on the basis of linear feedback (226). A second discrepancy, discussed in this section, concerns the timing of the yield in relation to the latency of reflex action. It will be

apparent from the data reviewed here that the compensatory signal begins too soon to be explained on the basis of an afferent detection of the onset of yield.

To consider the muscle yield first, the amplitude of stretch at which yield occurs increases with increasing stretch velocity; consequently, the time to yield does not decline simply in inverse proportion to stretch velocity. Recent measurements of the time to yield in the cat soleus (134) have shown that yield occurs at about 36 ms when stretch is applied at 10 mm/s, and it falls to about 18 ms for stretch velocities exceeding 30 mm/s. When the onset of reflex action is estimated from the point of departure between records of the reflex response and the underlying mechanical component, it is seen to be about 20 ms and to be largely independent of stretch velocity (134, 224). Under the same conditions, the onset of EMG change occurs approximately 10 ms after stretch begins. In other words, the onset of the EMG response that appears to be responsible for yield compensation arises about 10 ms before the yield could have occurred.

The arguments concerning latency of compensatory responses become even more forceful if one attempts to allow for the times for excitation and propagation of signals from spindle receptors and tendon organs. These times were measured in the same series of experiments in which the latency of yield and of reflex action were estimated (134). Typical minimum values, estimated from onset of a rapid stretch (100 mm/s) were as follows: activation of primary endings 5 ms, secondary endings 7 ms, and tendon organs 5 ms (provided that the tendon organ in question was above threshold). The addition of afferent conduction time (2-4 ms), central (spinal) delays (1-3 ms), efferent conduction time (2-3 ms), neuromuscular transmission (1 ms), and excitation-contraction coupling (5-10 ms) means that a minimum loop delay of perhaps 20 ms must intervene before events sensed by muscle proprioceptors can result in a compensatory force change. Since reflex action can begin as early as 20 ms after stretch onset, if this compensatory signal is based on a simple force-length comparison, the yield would have had to occur at or near stretch onset. Instead, the yield is delayed by at least 18 ms.

These considerations suggest that some form of prediction is involved in yield compensation. Furthermore the mechanism cannot simply be the use of time derivative elements in the feedback pathways, since a prediction based on the rate of force or length change would be of limited value in anticipating the occurrence of an abrupt event like muscle yield. Instead one can attribute several of the predictive features of the motor servo to nonlinearities in the response properties of primary endings. Dynamic responses to stretch are large and those to release are small (i.e., the asymmetry shown in Fig. 35B); this is well matched to the probability that stretch will cause a failure in stiffness whereas release will not. Stretch responses

rise rapidly to plateau values (differential sensitivity to small and large stretch), which insures a large, quick response that is well matched to the probability that there will soon be an abrupt failure in stiffness.

In the example shown in Figure 34 there is a close match between the time of yield and the onset of reflex action, but this is not always the case. At higher stretch velocities reflex action is delayed beyond the time of yield, whereas at lower velocities it may occur well in advance of yield (134, 226). Based on the kinematic data for cat locomotion reported by Goslow and colleagues (90), the 12.5-mm/s velocity in Figure 34 is intermediate within the physiological range. Although it is tempting to attribute this correspondence to evolutionary design, the idea is difficult to evaluate.

The latency of compensation depends on the length of the conduction pathway, which can vary appreciably between species and also between muscle groups in the same species (cf. ref. 187 for some values in humans). In contrast the latency of yield might not vary substantially if the percentage of length change associated with yield were an invariant feature (cf. *Instantaneous Stiffness and Short-Range Elasticity*, p. 272). However, one must also consider the extent to which relative stretch velocities vary with species size and with muscle group. The delay in yield associated with lower velocities in larger animals would help compensate for longer conduction times. On the other hand, more distal muscles are probably subjected to higher stretch velocities than more proximal ones, which would aggravate the problems associated with longer conduction pathways. One should not expect more than a rather rough match between time to yield and onset of reflex action. The low-pass filtering properties of the muscles and of inertial loads would tend to smooth out the deleterious effects of small discrepancies in timing.

It is apparent from the speculative nature of the previous discussion that more data comparing mechanical responses with reflex action would be helpful. What hinders this effort, particularly in experiments with intact subjects, is the difficulty in obtaining valid estimates of underlying mechanical components of response. One approach is to concentrate on force responses prior to the onset of reflex action, but even then the response is complicated by the presence of reaction forces associated with limb inertia and by onset oscillations in the stretching apparatus. The peculiar force transients at stretch onset reported previously (114, 206) undoubtedly contain inertial components, but they may also contain indications of muscle yield prior to reflex action. The force and EMG traces in Figure 36 show several phases associated with a subject using his arms to halt a free fall of his body. The subject was blindfolded and did not know when he would contact a supporting surface; thus the buildup of triceps EMG prior to contact (time zero) represents a programmed preparation of a postural

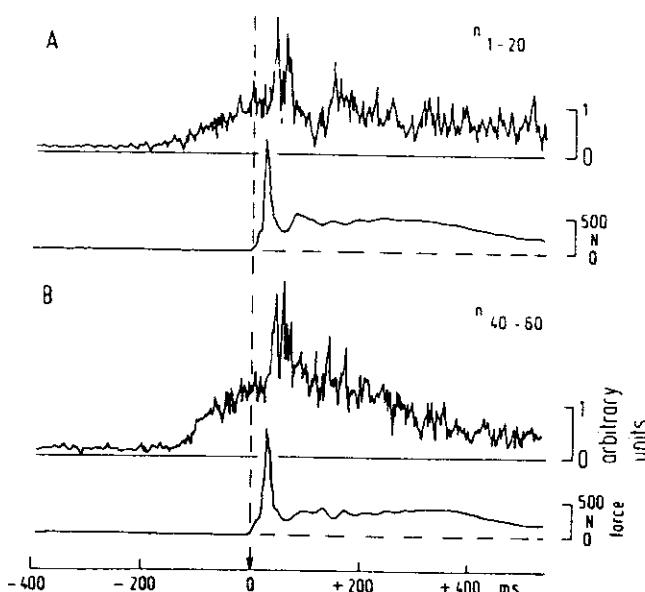


FIG. 36. EMG responses from left triceps brachii collected during a series of 60 falls, in which subject falls forward until movement is arrested by outstretched arms. Records are centered about the moment of impact. Upper traces in each panel are rectified EMG. Lower traces are of vertical force exerted against a platform. Panel A shows averaged responses to first 20 falls, Panel B, the response to the last 20 falls in the sequence. [From Dietz et al. (52).]

state. The rise in force after contact was gradual initially (finger and soft tissue contact) but became a sharp spike as the base of the hand impacted with the supporting surface. The spike undoubtedly contains an inertial reaction component, whereas the dip after this could conceivably represent muscle yield. If so, the timing of the EMG response is appropriate to prevent the dip from continuing and to establish a plateau level of supporting force.

The previous results appear to provide a highly relevant example of yield compensation, but the complexities in the interpretation of intact subject data are also apparent. Recent efforts to separate out inertial, muscle mechanical, and reflexive components of force development and stiffness (5, 164) provide tentative support for yield compensation and also suggest that we will soon be able to make much better comparisons between the easily controlled results obtained with functionally isolated muscles and the highly practical results obtained with intact subjects.

#### *Predictive Compensation: Feedforward vs. Nonlinear Feedback Viewpoints*

The analysis provided in the previous section suggests that compensation for yielding represents the action of a predictive mechanism, since the compensatory EMG signal is generated before the time at which an error in stiffness actually occurs (i.e., in preparation for the yield). In a more conventional type of feedback regulator the compensatory signal would

be produced by the error in stiffness and thus would occur only after the yield in muscle force had been detected by sensors such as tendon organs. The production of a compensatory signal in advance of the error is instead more typical of the actions of a feed-forward regulator as discussed in *Feedback, Feedforward, and Adaptive Systems*, p. 260.

The reflex pathway from spindle receptors does in fact operate in a feedforward mode under the conditions imposed in most experimental studies of functionally isolated muscles. This is because muscle length is rigidly controlled by the stretching apparatus, which opens the length-feedback loop. Muscle force is not allowed to influence its own length as it normally does by its direct action on the mechanical load (Fig. 1). Thus under certain experimental conditions spindle receptors function as feedforward sensors, whereas under most natural conditions they function as feedback sensors. The predictive mechanism is likely operative in both cases. Correspondingly it seems unwise to use the feedforward nomenclature, since this would stress operation under restricted experimental conditions, rather than operation under more natural circumstances. Rather, it seems wiser to attribute the predictive aspects of motor servo performance to nonlinear feedback, resulting from the presence of nonlinear sensors (primary endings) in the length-feedback pathway. In addition, we would point out that the term predictive compensation is not wedded to either the feedforward or the feedback viewpoint.

#### *Vibration and the Stretch Reflex*

The marked sensitivity of spindle primary endings to tendon vibration has been utilized as a tool to investigate the central actions of primary and, indirectly, secondary spindle afferents. These studies depend upon two useful attributes of vibration. First, provided that vibration is of sufficient amplitude and frequency, virtually every primary ending in the muscle will be discharging at the vibration frequency. Second, vibration effectively clamps the discharge of primary endings, making them unresponsive to static length change and largely unresponsive to superimposed constant velocity stretch.

The relation between vibration frequency and Ia-afferent discharge can be used to estimate the gain of the Ia-projection. For example, Matthews (197) reported that the force output of the TVR, measured in the soleus muscle of the decerebrate cat, increased in proportion to vibration frequency over a range of about 50–300 Hz. If one presumes that all of the primary endings in the muscle were discharging at the vibration frequency, the slope of this relation between frequency and reflex force provides an estimate of net Ia effectiveness. Matthews reported slopes ranging from 1.5 to 8.5 g/Hz with a mean of 4 g/Hz. Expressed in terms of afferent discharge, these units become g·impulse<sup>-1</sup>·s, measured over the total Ia-afferent pop-

uation of the muscle. Broadly comparable estimates were also reported by Kanda and Rymer (1977).

These estimates can be used to calculate primary ending contributions to the stretch reflex. For example, if one assumes a mean positional sensitivity of the primary ending of  $5 \text{ impulses} \cdot \text{s}^{-1} \cdot \text{mm}^{-1}$  (97, 135), then the primary ending contribution to static reflex stiffness is probably no more than 20 g/mm (approximately 0.2 N/mm). The stiffnesses of the stretch reflexes were typically about 100 g/mm (measured in the same preparations). Based on these calculations and the assumption that the residual stiffness (here approximately 80 g/mm) was induced by secondary spindle afferent projections, Matthews suggests that the secondary input may be as much as four times more potent than that derived from primary endings. While there are a number of reasons why this approach may underestimate the magnitude of Ia effects [see Matthews (202) for analysis], the Ia contribution is quite unlikely to be sufficient to account for the stretch reflex.

The assumption that the mechanical response of the muscle was not a significant source of static stiffness (202) may not be entirely accurate, although it probably was not a source of large-scale error (cf. ref. 102). The problem of uncertain mechanical contribution was circumvented by Kanda and Rymer (154), who used changes in static length and vibration of one muscle (the medial gastrocnemius) to examine reflex output in a synergist (the lateral gastrocnemius). Estimates of primary and secondary effects calculated in

this manner were closely comparable to those reported by Matthews (202), ranging from equal effect to five times greater secondary action.

The relative contributions of the reflex and mechanical components can also be compared readily by including an assessment of mechanical response in the analysis. Figure 37, derived from a study using the soleus muscle of the decerebrate cat (134), compares the stretch response superimposed upon an established TVR with the stretch reflex initiated from a comparable initial force. There is also included an estimate of the muscle mechanical response.

The superposition of reflex, vibration, and mechanical records highlights several important differences between the responses. Clearly the mechanical contribution to static force is negligible at the initial length used here, supporting Matthews contention that mechanical properties do not account for the (residual) stiffness previously described. Also, superimposed vibration has suppressed almost all of the initial dynamic force increase of the stretch reflex—in fact, the resulting force transient looks rather like the underlying muscle response. In spite of the rough similarity of muscle and reflex response in the initial phase, the later portion of the vibration response diverges sharply from the mechanical record and approaches the magnitude of the full stretch reflex. This latter divergence suggests that some additional source of excitation is being introduced by muscle stretch. Jack and Roberts (147) argued that such excitation may result from more secure entrainment of primary ending discharge

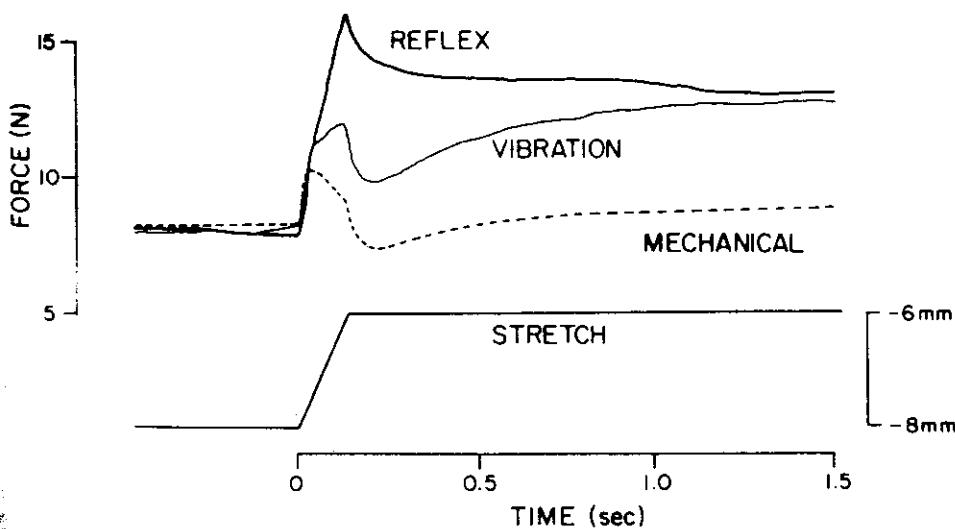


FIG. 37. Vibration of the soleus muscle in the decerebrate cat prevents reflex compensation for muscle yield. Longitudinal tendon vibration was used to block the stretch response of primary endings to demonstrate the importance of the dynamic response in the transient phase of stiffness regulation. The initial force of the normal reflex response was augmented to match that developed in the presence of vibration with a cross-extensor reflex. The difference between reflex and vibration *traces* is attributed mainly to primary endings, whereas the difference between vibration and mechanical *traces* is attributed mainly to secondary endings, although tendon organ inhibition may limit the magnitude of this component. Mechanical *trace* is an estimate, based on stretch of muscle stimulated electrically at 8 pulses/s via the ventral roots. Reflex and vibration responses are ensemble averages. [From Houk, Rymer, and Crago (137).]

at the longer muscle length; however, the relative similarity of the initial mechanical and vibration force transients indicates that practically all the primary endings were entrained at the initial length. The additional excitation induced by stretch was quite probably caused by the activation of some other stretch-sensitive receptor, such as the secondary endings. Moreover the close similarity of stretch reflex and vibration response indicates that this secondary ending contribution was probably very potent and may be largely responsible for the static stiffness. Finally, while the late increase in vibration response shown in Figure 37 was not always large in other preparations (134), it was almost always larger than the mechanical component, suggesting that a secondary contribution was uniformly present.

The experiment combining vibration and stretch also provides evidence supporting a predictive or anticipatory role for the primary ending in preventing muscle yield during stretch. Specifically Figure 37 also shows that vibration suppressed virtually all of the initial dynamic force increase in the stretch reflex leaving a residual force transient rather like the yield of the mechanical response. Recordings from spindle primary endings during vibration and stretch (134) reveal that the primary dynamic response is largely occluded by the vibration. Although the primary ending may respond with more than one impulse for each vibration cycle during large amplitude dynamic stretch (204), this effect is usually relatively modest. Vibration had virtually no effect on secondary endings, so these endings are unlikely to be involved; however, tendon organs often show variable force-dependent phase-locking of discharge (28). Augmented tendon organ discharge may have caused the reflex suppression; however, evidence cited earlier (*Loop Gain of Force Feedback*, p. 288) suggests that the central actions of Ib-afferents are modest in the decerebrate preparation. The yield in force during vibration more likely arises because the dynamic response of the primary ending has been essentially eliminated. Preservation of the dynamic response of the primary ending is associated with yield compensation, while an occlusion of the dynamic response allows yield to take place. Apparently the dynamic response is being used to compensate for yield in an anticipatory manner.

#### *Velocity Dependence and Damping*

To this point, we have emphasized the springlike behavior of the motor servo. However, given that muscles act upon substantial inertial loads, and given the existence of significant loop delays, the damping properties of motor servo action are likely to be important in preventing oscillation and instability. Oscillatory behavior in man-made mass-spring systems is often minimized by viscous damping, in which the damping element produces a restoring force proportional to movement velocity. This would require that

dynamic force output of the motor servo exceed the static force and that the dynamic increase be proportional to movement velocity. With this analogy in mind, it is instructive to examine velocity sensitivity of the motor servo response.

The velocity sensitivity appears to be sharply different for small- versus large-amplitude movements. Small-amplitude responses have been examined using sinusoidal length changes, in which the frequency of the applied sinusoid can be equated broadly to the velocity of the ramp stretch. As we have described earlier in *Dynamic Features of Force Development*, p. 297, under these conditions the primary ending shows linear velocity dependence above a corner frequency of about 2 Hz; and this velocity dependence is apparently transmitted to the motor output without evident modification in frequency response. These features, which are analogous to those of a high-pass filter, are well matched to compensate for the low-pass features of muscle, resulting in a net flat frequency response relation (242). In sum, reflex force output shows essentially no dependence on velocity in the small-signal region.

For larger stretches, velocity sensitivity is most readily assessed by measuring the force produced at a particular amplitude of ramp stretch, over a wide range of stretch velocities. Figure 38, derived from a study performed on the functionally isolated soleus in the decerebrate cat (132), shows that the force increase that occurs with increasing stretch velocity is rather modest—a 100-fold increase in velocity is accompanied by less than a 2-fold increase in reflex muscle force. The force increases are largely confined to slow stretch velocities, less than 5 mm/s (226). Similar modest velocity dependence has been reported in a number of other studies on isolated animal muscles that used either constant velocity stretches (193, 270) or sinusoidal movements (150, 171, 252, 254). There is also comparable modest velocity dependence evident in the response of the human long thumb flexor to ramp stretch covering a wide range of velocities (257). In no case was the force increase simply proportional to the change in velocity.

When plotted on linear coordinates, the relation between force increment and velocity for the functionally isolated soleus is a negatively accelerating curve (226), which indicates that a power or logarithmic function might best fit the data. In a recent study of soleus reflex response to ramp stretch (136), the relation between the log of dynamic force increment and log of stretch velocity was shown to be well fitted by a straight line with a slope of 0.2. A straight-line relation on log-log coordinates implies that a power function (here with fractional exponent 0.2) would best describe the velocity dependence of motor servo output during constant velocity stretch. Similar exponent values were shown by plots of log EMG increment versus log velocity. Overall, the force increase recorded with increasing velocity is quite modest and

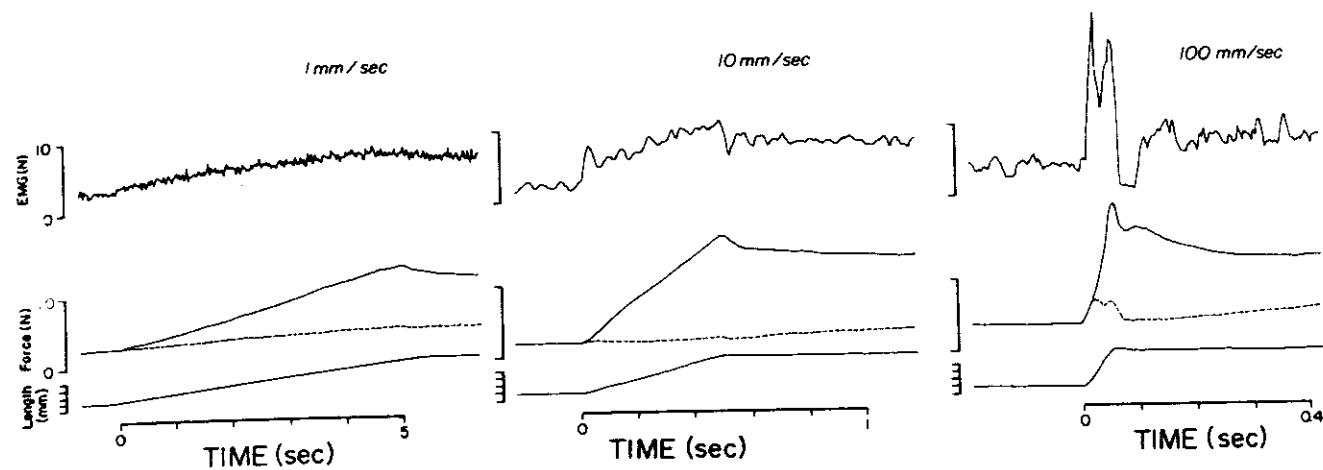


FIG. 38. Lack of dependence of EMG and force of the soleus on stretch velocity. Records show averaged response to 10 mm stretch, applied at three velocities, 1, 10, and 100 mm/s. Force output, measured at ramp end, shows approximately a 2-fold increase for a 100-fold increase in stretch velocity. EMG increment measured at the same point is slightly larger, but increase is still modest in comparison with increase in velocity. Dashed traces represent the mechanical responses. [From Houk (132).]

has a character quite different from that shown by a linear damping element.

There are a number of parallels between the velocity dependence of motor servo output and the velocity dependence of spindle receptors assessed during ramp stretch that suggest that spindle receptors are the major contributing mechanism. For example, when the increment in discharge of primary and secondary endings is measured at a constant stretch amplitude, over a wide range of stretch velocities, the relation between rate increment and velocity is linear on log-log coordinates, with slope of 0.2–0.4 (137, 258). In analogy with the discussion of the previous paragraph, this relation implies that the rate increment is a fractional power function of velocity. It is perhaps of further interest that the velocity exponents of primary and secondary endings are essentially comparable, which implies that either (or both) could be responsible for the velocity dependence of the motor output and that neither type of receptor, alone or in combination, would promote simple linear viscous damping.

While the lack of simple velocity dependence implies that the motor servo does not provide linear damping, there may be a tendency for damping effects to be more prevalent at low stretch velocities, where force increases more steeply with increasing velocity. This type of response is perhaps more akin to static friction than to viscosity, in that the response is sensitive to the presence of movement but not very sensitive to the particular velocity at which the movement takes place. While the functional advantage of a system showing such frictional properties remains to be determined, it would seem likely that this type of feedback would resist the onset of motion of inertial systems (when movement amplitudes and velocities are invariably small) but would be relatively unimportant for larger, more rapid movements.

### Summary

Comparison of parametric dependencies of stretch reflexes with corresponding dynamic properties of areflexive muscle and muscle proprioceptors provides helpful insight concerning motor servo function. The dependence on initial force suggests that as force increases there is a progressive shift from a reflexive to a mechanical dominance. Dynamic incremental stiffness is less dependent on initial force than is the underlying muscle mechanical component, which is similar to the results for static stiffness discussed in *Dependence of Incremental Stiffness on Initial Force*, p. 287. Comparison of reflex and mechanical dependencies on amplitude demonstrates a marked improvement in linearity that results from reflex action. Nevertheless, reflexes may show 2–4 times greater stiffness for small stretches (about 1% of physiological range) than for larger ones. This difference is attributable to mechanical properties in the dynamic phase and probably to the differential sensitivity of primary endings in the static phase.

Comparison of responses to stretch versus release reveals marked asymmetry of reflex action. The greater increase in motor output during stretch (as contrasted with decrease during release) is attributable to a similar asymmetry in the dynamic responses of primary endings. Since the mechanical components show an opposite asymmetry, it appears that asymmetric reflex action serves as an important compensatory mechanism contributing to stiffness regulation. Reflex action begins too soon after stretch onset to be accounted for by a linear force-length comparator model. Instead, it is attributable to nonlinear sensor properties of primary endings. These properties result in predictive compensation, since they insure that a large reflex is initiated shortly after stretch onset. This

reflex is effective in compensating for the yield in muscle mechanical force that does not occur until several milliseconds later.

The results obtained by using vibration to clamp and control primary ending discharge help to define the relative importance of primary and secondary endings. The large dynamic responses of primary endings are particularly important under dynamic conditions in maintaining motor servo stiffness during stretch. Responses of secondary endings probably are especially important in regulating static stiffness.

For small-amplitude stretches motor output shows a proportional dependence on velocity, which derives from the properties of spindle receptors and compensates for the low-pass characteristics of muscle. With intermediate to large stretch amplitudes, however, motor output and reflex force both show fractional power dependencies on velocity that derive from similar properties of spindle receptors and of a reflexive muscle. This results in a frictionlike force opposing motion that may be advantageous in providing damping at low velocities without greatly slowing large and rapid movements.

#### IMPLEMENTATION OF MOVEMENT COMMANDS

To this point, we have concerned ourselves largely with the problem of motor servo regulation, which is to say that the reference signal for the motor servo is unchanging. Under these conditions, applied perturbations in load force or in position cause the system to traverse the characteristic force-length relations described in earlier sections. A rather different outcome arises if the reference signal is varied intentionally, in which case it is called a command signal. In this section, we describe how such variations in the reference signal give rise to changes in the locus of static and dynamic force-length curves. Depending on the characteristics of the load, these locus transitions usually induce a state of disequilibrium, which gives rise to movement.

Before we can consider precisely how variations in the command signal may give rise to movement, we must review the ways in which the motor commands are distributed to the key efferent elements— $\alpha$ -,  $\beta$ -, and  $\gamma$ -motoneurons. We also review the ways in which the afferent signals, especially those derived from spindle receptors, interact with and modify the discharge of motoneurons during movement.

#### $\alpha$ - $\gamma$ -Relations

The efferent innervation of the muscle spindle has been described in detail in the chapter by Matthews in this *Handbook*. In this section, we focus on those aspects of the efferent innervation of extrafusal and intrafusal muscle that are most relevant to the movement control hypotheses discussed earlier (Figs. 7–9; see also *Spindle Receptors as . . . Detectors*, p. 265,

*Conditional Feedback and Servo Assistance*, p. 265, and  *$\beta$ -System and . . . Zero Sensitivity*, p. 266). Recordings from spindle receptor afferents during isometric contraction of human forearm (278–280, 282, 283) and leg muscles (29–32), from hindlimb muscle afferents during locomotion of the cat (104, 174, 246, 247, 262), and from jaw-closing muscles of the monkey (176) have shown that spindle afferent discharge rate increases with the onset of muscle activity. Simultaneous excitation of spindle receptor afferents and muscle is also evident during slow shortening of flexor muscles in the cat hindlimb, (174, 246) and during slow shortening of human finger flexors (281). These findings have been uniformly interpreted as implying joint activation or coactivation of  $\alpha$ - and  $\gamma$ -motoneurons (*Spindle Receptors as . . . Detectors*, p. 265).

In some studies coactivation has been associated with increases in spindle receptor discharge that parallel increasing force over a considerable range (32, 283). This covariation has been interpreted to imply that increases in fusimotor discharge rate and in fusimotor recruitment continue up to high levels of motor output. Moreover abrupt discontinuities in spindle afferent discharge (32), which occur over a range of force levels, have been attributed to recruitment of  $\gamma$ -motoneurons.

In contrast a number of studies report evidence that increased spindle afferent discharge and muscle contraction may not uniformly coexist. For example, spindle afferent discharge may decline or cease completely during shortening of cat jaw muscles (41) or of contracting hamstring muscles of the running cat preparation (174, 246). In the falling cat, spindle afferent discharge in hindlimb muscles may also decline without obvious external shortening of the active muscle (247). It has also been reported that the threshold for activation of spindle receptors in human pretibial muscles may be varied independently of changes in skeleto-motor output (34).

There is also evidence that spindle receptors may be activated by efferent command in the absence of muscle excitation. For example, recordings in hindlimb muscles of the decerebrate cat (243, 260, 291) have shown that activation of muscle by the crossed extensor and other reflexes consistently gives rise to acceleration of spindle afferent discharge at stimulus intensities insufficient to activate  $\alpha$ -motoneurons. Furthermore recordings from afferents in jaw-closing muscles of the monkey (176) and tranquilized cat (272) show that spindle afferent discharge characteristically increases before the onset of EMG activity. It follows that the lower threshold for activation of spindle afferent fibers (and therefore fusimotor neurons) may not depend upon the particular preparation, but it may simply be a consequence of differences in the experimental arrangement. Although joint excitation of spindle receptors and of muscle is common, it is not always the rule.

A number of major unresolved problems exist in relation to the control of intrafusal and extrafusal innervation. The first and perhaps the major problem concerns the complexity of the control mechanism. One proposition holds that the presence of independent innervation of  $\alpha$ - and  $\gamma$ -motoneurons implies that functionally independent control mechanisms necessarily exist (34, 98) and that they are likely to be utilized in the course of movement control. This argument has been extended to include the proposition that static and dynamic fusimotor neurons may also be subject to independent control. The alternative position is that the command signals to  $\alpha$ - and  $\gamma$ -neurons are essentially coupled and that independent activation of these neuronal elements does not normally arise (cf. ref. 284). A second problem, largely unexplored at present, is that augmentation in spindle afferent discharge may not be due solely to  $\gamma$ -innervation— $\beta$ -fibers cannot now be ignored. The role of  $\beta$ -fibers is addressed in the next section.

The argument for independent activation of  $\alpha$ - and  $\gamma$ -motoneurons resides partly in the observation that, during some forms of movement, EMG activity may be present in a muscle from which no spindle afferent discharge is being recorded. Based on the cited examples it seems that these observations have often been made under circumstances in which the shortening velocity of the muscle was substantial and in which the effects of concurrent fusimotor activation may have been overwhelmed by the rapid muscle shortening (4).

The other argument advanced in support of independent control of  $\alpha$ - and  $\gamma$ -motoneurons depends upon the finding of spindle receptor activation in the absence of  $\alpha$ -motoneuron activity. Here, increased spindle afferent discharge has been uniformly attributed to  $\gamma$ -fiber activation. While the spindle afferent recordings in humans do not show afferent rate increases without EMG activity (111, 112, 284), separate activation of muscle and spindle receptor afferents has been clearly demonstrated in animals, as described in earlier paragraphs. These findings suggest that the control of  $\alpha$ - and  $\gamma$ -motoneurons may differ somewhat in human and animal subjects.

While it is conceivable that these results reflect fundamental differences in the patterns of activation of  $\alpha$ - and  $\gamma$ -motoneurons, there are also differences in the experimental arrangement that may be at least partly responsible for the apparent differences in response. One difference is the magnitude of series compliance, especially that provided by the soft tissue present at the point of apposition of a limb with some measuring device. There is no comparable compliance in functionally isolated muscles, or in jaw muscle preparations, since the teeth form a noncompliant link. The practical outcome of this compliance is that during simultaneous and rapid excitation of  $\alpha$ - and  $\gamma$ -motoneurons, the earlier onset of force in extrafusal muscle (which arises from more rapid conduction in

skeletomotor axons) may limit the development of tension in intrafusal fibers by inducing internal muscle fiber shortening (260). This shortening will arise normally from lengthening of series elastic elements but is likely to be considerably augmented in the presence of a substantial soft tissue compliance.

It is evident that in contracting muscle the interaction between muscle and the muscle spindle is potentially quite complex. The problem of assessing fusimotor effects in spindle afferent responses recorded during muscular contraction can be circumvented by recording directly from  $\gamma$ -motoneurons in conscious, behaving animals. The technical difficulty of this exercise has limited the number of reported studies; however, during isometric contraction of jaw-closing muscles in monkey (176) and cat (272), the fusimotor neurons are activated without any measurable force and EMG activity. In view of these findings, the apparent similarity of threshold for activating  $\alpha$ - and  $\gamma$ -motoneurons in human muscle may be related partly to the effects of series compliance and to the fact that voluntary activation of muscle in humans appears to induce a virtually simultaneous activation of  $\alpha$ - and  $\gamma$ -motoneuron pools. While these factors may help explain the differing patterns of response, it is also possible that CNS control of  $\gamma$ -motoneurons is different in human muscles, a possibility that is supported by the rather low resting discharge rate recorded in human muscle spindle afferents.

The possibility of independent control of  $\gamma$ -static and  $\gamma$ -dynamic neurons may be questioned. While there is now strong evidence that electrical stimulation of different regions of the CNS appears to selectively excite  $\gamma$ -static and  $\gamma$ -dynamic neurons (cf. ref. 220), this finding does not automatically imply that these pathways are subject to independent control under physiologically relevant conditions. This is because many regions of the nervous system would normally be coactivated in the course of voluntary movement.

#### *Positional Stiffness Deduced From Spindle Relations*

In previous sections, the potential contribution of  $\alpha$ - and  $\gamma$ -motoneuron excitation to motor output was discussed in relation to several models of motor servo action, such as the follow-up length servo arrangement of Merton (211) and the servo-assistance approach of Matthews (198). These models were derived largely without data on spindle afferent discharge or motor output in intact preparations. The availability of recordings from human nerves has provided descriptions of the quantitative relations between spindle receptor discharge rate and force in the same muscle, allowing these hypotheses to be investigated. For example, these relations have now been used to estimate the gain of a follow-up servo configuration in terms of the positional stiffness of the motor servo.

Using the finger flexors at the metacarpophalangeal

joint, Vallbo (283) estimated that the positional stiffness of a follow-up servo configuration was of the order of 0.03 Nm/deg. This stiffness calculation results in an upper bound since it is based on the assumption that the  $\gamma$ -motoneuron pathway is the sole source of command. Stiffness was calculated as the product of the positional sensitivity of spindle receptors (in pulses  $s^{-1} \cdot deg^{-1}$ ) and the torque-rate relation (in Nm  $pulse^{-1} \cdot s$ ), the latter being used as an upper bound of spindle afferent capacity to generate force. The resultant positional stiffness estimate (in Nm/deg) was deemed to be rather low, on the grounds that a modest load, such as 1 Nm applied at the finger tip would induce a 37° deflection at the metacarpophalangeal joint.

While the occurrence of an angular deflection of this magnitude might appear to indicate that positional stiffness, and thus length servo action, was inadequate, this analysis is somewhat arbitrary. A more objective assessment could depend upon a comparison of the normalized positional stiffness of the servo with that of the muscle, on the premise that an effective position servo should augment muscular stiffness by a substantial margin. The maximum torque output of the finger flexor is about 4 Nm, and the range of motion at the joint is 100°. Using the formulation from section *Normalized Stiffness*, p. 285, to estimate stiffness, the calculation provides a normalized value of 0.75, which can be compared with the 2.0 upper bound for the normalized mechanical stiffness of skeletal muscle provided in *Mechanically and Neurally Mediated Components*, p. 285. The resultant conclusion that motor servo stiffness is likely to be less than maximal muscle mechanical stiffness constitutes a strong argument against the hypothesis for length regulation.

#### $\beta$ -Innervation of Muscle Spindles

Acceleration of spindle afferent discharge in contracting isometric muscle has usually been attributed to  $\gamma$ -motoneuron activity. While increases in discharge rate that arise in the absence of EMG activity are legitimately attributed to fusimotor action, increases arising above extrafusal threshold could arise equally from  $\beta$ -fiber action.

Skeletofusimotor or  $\beta$ -fibers are simply large-diameter motor axons that branch to innervate both intrafusal and extrafusal muscle fibers (see the chapter by Matthews in this *Handbook*). Their existence was first recognized in frog muscle (155) and they have since been identified in the muscles of many reptiles (64, 167, 220). Although  $\beta$ -fibers were also isolated in several mammalian muscles more than 15 years ago (1, 22, 23), these muscles were small (superficial and deep lumbricals), and their innervation was thought to be nonrepresentative. The importance of this innervation was also discounted on the premise that the  $\beta$ -innervation was simply a vestigial remnant of the form of innervation that prevails in more primitive species.

Based upon the use of more sensitive techniques (14, 15, 63, 115, 148),  $\beta$ -fiber innervation of muscle spindles has been reported in 30%–70% of mammalian muscles examined (63, 205) in several different species (64, 220), and it is now evident that their significance must be seriously reevaluated.

At this time, there is relatively little information available regarding possible functional contributions of  $\beta$ -motoneurons in physiologically activated muscles. Post, Rymer, and Hasan (243) have reported evidence derived from triceps surae muscles in the decerebrate cat, suggesting that the augmentation of spindle receptor afferent discharge that arises during reflexively induced isometric muscle contraction may be due largely to the activation of  $\beta$ -axons. This hypothesis was based, in turn, on the finding that the range of recruitment and rate modulation of  $\gamma$ -motoneurons was apparently quite limited in this preparation, so that both  $\gamma$ -motoneuron discharge rate and the capacity to recruit fresh  $\gamma$ -motoneurons was apparently saturated near the point of skeletomotor threshold. More recently, the use of ensemble averages of EMG that are synchronized to the spindle afferent spike has provided preliminary evidence that  $\beta$ -innervation may be detected by finding correlated unitary EMG activity prior to the spike occurrence (260). This EMG activity, which precedes the spike by some 5–10 ms, is the expected outcome if  $\beta$ -motoneuronal discharge was able to excite both intrafusal and extrafusal muscle. Nonneural interactions, however, arising from the mechanical effects of contraction of adjacent motor units could conceivably give rise to similar effects, and the technique requires further evaluation. In any event, it seems likely that  $\beta$ -motoneuron activity may serve to augment spindle receptor discharge above extrafusal threshold, while for animal models at least,  $\gamma$ -motoneurons may be much more influential at subthreshold levels of spinal excitation.

#### Analytical Approaches to Actions of $\alpha$ -, $\beta$ -, and $\gamma$ -Motoneurons

It is now instructive to reevaluate the observations of spindle afferent discharge from the various preparations in relation to the analytical models that were outlined in *CONTROL THEORY CONCEPTS*, p. 258, and *HYPOTHESES OF MOTOR SERVO FUNCTION*, p. 264, and in Figure 7. A potentially productive approach, described in these sections, is to treat the arrangement of extrafusal and intrafusal muscle as the equivalent of a model reference system, in which motor output is distributed to both the controlled system (the extrafusal muscle) and to a model of this system (the intrafusal fiber). If a command induces changes in the controlled system that are precisely comparable to the changes in the model, then no error signal will result. In the context of the motor servo, spindle afferents would produce no alteration in discharge under conditions in which length changes of the intrafusal fiber

exactly match those of the extrafusal fibers. (The length under consideration here is the length of the contractile portions of the spindle poles. Since both intrafusal and extrafusal muscle fibers insert into the same common tendon, the external length of the system must be the same for both elements.) During command-induced excitation of the system, feedback is conditional, because it arises only under the conditions in which performance of the controlled system and of the model differ.

In relation to the spindle afferent recordings reported earlier, one possible objection to a model reference configuration is that  $\gamma$ -efferent discharge does not appear to vary in any simple relation with skeleto-motor activity. Specifically, if  $\gamma$ -motoneuron output is not appreciably modulated in parallel with increasing  $\alpha$ -motoneuron output, as suggested in the previous section, the nervous system might not be able to take advantage of the model reference arrangement. However, if  $\beta$ -fibers turn out to be an effective source of excitation to intrafusal fibers, then the  $\beta$ -motoneurons might extend the dynamic range of efferent innervation of the muscle spindle and allow a model reference arrangement to be implemented.

As was pointed out in **HYPOTHESES OF MOTOR SERVO FUNCTION**, p. 264, conditional feedback from spindle receptors does not presume absent afferent discharge; rather it requires that spindle receptor discharge, under appropriate loading conditions, be maintained at

the constant discharge rate. There are relatively few reported circumstances in which spindle afferent discharge is unchanging in the face of command-induced changes in motor output. In the contraction of isometric muscle for example, there is characteristically an augmented discharge rate, which indicates that intrafusal tension typically rises more quickly than can be offset by force-related internal shortening. Similarly, during command-induced free shortening of muscle, as seen in human leg muscles (113) and in monkey (87) and cat jaw muscles (41), there is often a reduction in spindle afferent discharge. On the other hand, during very slow voluntary shortening of human finger flexors (281) or during flexion of hamstring muscles in the walking, intact cat (244) or jaw-closing muscles in the monkey (87), spindle afferent discharge may remain relatively constant. This latter situation presumably represents the null condition discussed in *Spindle Receptors as Model-Reference Error Detectors*, p. 265.

Overall, these findings indicate that in many naturally occurring circumstances, the properties of the intrafusal muscle are not consistently well matched to those of the extrafusal muscle and load. The major reason for a difference in behavior of the controlled system and of the model may be the dependence of the extrafusal system on load. While intrafusal and extrafusal muscle fibers might behave similarly under isolated conditions, the presence of a load will modify the response of extrafusal muscle to imposed com-

mand. The properties of the two systems might be well matched, for example, when the load is simply equal to the inertia of the limb.

An alternative arrangement, which is insensitive to load, was spelled out in *Control Configurations*, p. 262. This is the zero-sensitivity configuration, in which the difference between model and system response, the reference error, is fed back to the model to form a positive-feedback loop (Fig. 7A, option 2). We argued in  *$\beta$ -System and ... Zero Sensitivity*, p. 266, that this configuration is analogous to the  $\beta$ -arrangement, in which the reference error (spindle afferent discharge) is fed back onto the system model (intrafusal fiber) via an excitatory synaptic projection to  $\beta$ -motoneurons. Under the conditions in which the gain of the positive-feedback loop is 1, it can be shown (127) that the system performance should become independent of the properties of both extrafusal muscle and load and dependent solely upon the properties of the feedback pathway.

To our knowledge, there is no evidence that bears directly on the presence of a zero-sensitivity configuration in the motor servo; however, a number of related issues should be mentioned. It has been claimed that the action of  $\beta$ -fibers, as determined by their capacity to modulate spindle afferent discharge, is modest. If correct, this would imply a remote prospect of significant loop gain in the positive-feedback loop. On the other hand,  $\beta$ -effects have nearly always been examined in isometric muscles, in which  $\beta$ -dynamic effects might not be readily discernible. Moreover, the effects of  $\beta$ -axon stimulation have usually been examined in muscles devoid of fusimotor activity, which may have meant that the background tension of the intrafusal fiber was unusually low. Although no evidence of a positive-feedback configuration exists in the mammal, findings supporting the existence of such a loop have been reported in the frog preparation, in which spindle receptor response to stretch fell after section of the dorsal roots (144).

#### *Equilibrium Point Control*

The results just reviewed indicate that the relations between  $\alpha$ -,  $\beta$ -, and  $\gamma$ -motoneurons are not fully understood at the present time, although considerable progress is being made. One might suppose that this lack of understanding would pose a major obstacle in attempts to understand movement control, but, in fact, considerable progress has been made. This success appears to be related to the finding discussed in *Actions of Control Signals on the Motor Servo*, p. 286, which is that the basic effect of a central motor command is a shift in the threshold of the stretch reflex, more or less independent of the  $\alpha$ - and  $\gamma$ -motoneuron makeup of the command. Thus the salient properties of the motor servo are well summarized by force-length or torque-angle relations, which can be measured directly even though they derive from complex

physiological mechanisms (TONIC STRETCH REFLEX IN FUNCTIONALLY ISOLATED MUSCLES, p. 283, and STATIC REGULATORY CHARACTERISTICS IN INTACT SUBJECTS, p. 290). Movement problems can then be understood on the basis of an interaction between one of these summary relations and the relevant mechanical load. This approach was introduced in *Summary Model*, p. 268 (cf. Figs. 11 and 12). It will be helpful to elaborate on a simple example given there that is relevant to a functionally isolated muscle before proceeding to more complex examples that deal with the elbow musculature as a whole.

Trajectory b in Figure 11 shows the hypothetical result of instructing a functionally isolated muscle to shorten by pulling on a spring that is attached to it. The movement begins from an initial postural state (filled circle) that lies long the solid force-length curve representing the properties of the stretch reflex at the initial value of central motor command. The command to move is assumed to be a step change in the value of the central motor command, which shifts the threshold of the stretch reflex to the left. Correspondingly the entire force-length relation is shifted to the left (dashed curve). This causes muscle force to rise, and as it rises the spring load is stretched while the muscle is shortened. Note that the new muscle force and length finally achieved depend on the spring constant of the load (the slope of trajectory b). The same command would result in less shortening, but more increase in muscle force, if the spring constant of the mechanical load were greater. In more general terms, the example illustrates why the actual movement produced by a given motor command is appreciably influenced by the mechanical load. It is equally clear that the movement is influenced by the stiffness of the stretch reflex. If motor servo stiffness were greater (steeper slope of dashed curve), the amount of shortening against a given spring load would also be greater. Figure 12 helps to emphasize this incessant loop of interaction between the stiffness property of the motor servo ( $\phi$ ) and the properties of the load (L).

The same basic approach can be used to analyze movements of the elbow using the torque-angle relations that have been measured for the elbow joint. The reader will recall from *Torque-Angle Relations*, p. 292, that the solid lines in Figure 31 represent the equivalent stretch reflexes of all the elbow flexor and extensor muscles acting in combination at each of three different levels of central motor command. The dashed curves are load lines describing the mechanical load in terms of the torque required at different elbow positions to support each of several different weights. The initial postural state of the elbow is specified by its angle together with the torque it exerts to counteract the mechanical load. Thus, any given equilibrium point in Figure 31 characterizes a postural state, and movement can be treated as a transition in equilibrium that occurs as a consequence of a shift in the torque-angle curve caused by a change in the level of central

motor command. For example, if the central command is initially at level (2) and the load is at level 3, the initial posture is specified by the intersection at  $\alpha_3^{(2)}$ . A change in central command to level (1) would result in a transition to a new postural state specified by the intersection at  $\alpha_3^{(1)}$ , a movement to a more flexed position at which the musculature produces a slightly lower torque.

The previous examples suggest that the CNS controls movements by setting central motor commands to particular constant values and by then letting the particular modulations in torque required to execute the movement evolve as a natural consequence of mechanical and reflex interactions at the level of the motor servo. By this hypothesis errors in the selection of appropriate central motor commands are not corrected on any continuous basis; instead, error correction at this higher level is assumed to occur only as a result of discrete reaction-time movements, as in Figure 13.

Good evidence in support of this hypothesis has come from experiments in which unexpected load changes have been applied during occasional trials of well-rehearsed movements. Fel'dman (73), who studied human elbow flexion movements triggered by an auditory cue, suppressed corrective reaction-time movements by eliminating visual feedback and by asking his subjects not to intervene voluntarily. Bizzi, Polit, and collaborators (25, 26, 238) studied both head and elbow movements in primate subjects cued by a visual display; they suppressed corrective movements by eliminating visual (and sometimes other modalities) feedback during the movement. More recently Kelso and Holt (156) studied finger movements under conditions analogous to Fel'dman's. In all three sets of experiments it was shown that the final postural state was not affected by transient mechanical disturbances, ones that were sufficiently large to have caused a substantial somatosensory barrage of activity. Disturbances applied during the latent period prior to movement altered the initial limb position, and disturbances applied during the movement produced major changes in joint trajectory, but in both cases the joint angle and torque promptly settled to the same values as obtained during undisturbed control movements. In contrast the application of steady loads did disturb the final postural state, but, as shown by Fel'dman (72), the observed alterations in angle and torque were precisely the ones predicted from the stereotyped torque-angle curves described earlier and therefore did not involve changes in central motor command.

It has also been shown that interference with sensory feedback does not alter this basic result. Rhizotomized monkeys (26, 238) and human subjects with pressure blocks (156) still make movements to final positions that are not appreciably altered by transient mechanical disturbances. The absence of any major deterioration in performance probably derives from the gross similarity between the length-tension rela-

tions of a reflexive muscle and the force-length relations of the intact motor servo as discussed earlier (*Mechanically and Neurally Mediated Components*, p. 285). In addition, the results indicated that the subjects often employed unusually high levels of co-contraction, presumably as a mechanism for stiffening the musculature in compensation for the absence of stretch reflexes. The latter findings suggest a shift in strategy following sensory deprivation, although it should be noted that intact subjects may also employ cocontraction during the execution of voluntary movements (74, 75). The major impact of these results, however, is to reinforce one's confidence in the notion that movements do not require a continual updating of central motor commands.

Neurologists have long debated whether or not it is proper to treat movement as a transition in posture (123, 192). The view of Holmes, who supported this idea, appears to be quite consistent with the concept of equilibrium point control. According to this concept, the CNS need only select new levels for the central motor commands sent to the motor servos controlling the body's musculature. The subsequent result, mediated by autogenetic reflexes and muscle mechanical properties, should be a smooth transition from one postural state to another. Although this explanation does not deal with the question of controlling how rapidly the transition is executed, the latter feature may be partly explained in terms of simple extensions of the equilibrium point concept. For example, slow movements could be produced by progressive shifts in reflex thresholds as contrasted with the stepwise shifts implied in the earlier discussion. Furthermore movements could be speeded up by producing an initial threshold shift that is larger than necessary, followed by a return to an appropriate static level. This would amount to a pulse-step command of the type known to control eye movements (253) and recently suggested for limb movements as well (81, 84, 85). It is clear that equilibrium point control should be given serious consideration since it is potentially capable of controlling quite general motor functions.

#### *Stiffness Regulation vs. Stiffness Control*

Stiffness regulation is defined as a preservation of a relative constancy of reflex stiffness, whereas stiffness control implies centrally induced changes in this property, produced to achieve some end such as the stiffening of a joint or the production of a movement. At several points throughout this chapter, evidence has been provided that the pattern of motor servo action is one that tends to reduce variations in stiffness. For example, the greater reflex action at low vs. high initial forces compensates for the proportional dependence of muscle mechanical stiffness on initial force, and the characteristic asymmetry of reflex action compensates for an opposite asymmetry of mechanical responses. While these observations provide support for the no-

tion of stiffness regulation, there is no implication of stiffness control. In fact, it has already been pointed out that attempts to get subjects to control the stiffness of their musculature have met with only limited success (*Effect of Instructional Set*, p. 294, *Gain Variation vs. Gain Control*, p. 296). Stiffness is clearly increased when the musculature is brought from a relaxed to an active state, but once an activity pattern is set, there appears to be little additional capability for modulating gain to control stiffness, at least not in preparation for disturbances in posture. Another possibility, recently explored by Cooke (44) and by Sakitt (260a), is that stiffness control is used as a mechanism for making movement.

In Figure 39, *part A* shows tracking movements of a human subject and *part B* shows that analogous output from a model in which movements are produced by controlling stiffness, as elaborated in *part C*. The solid lines show force-length relations for an agonist muscle in the presence of three different central motor commands. The assumption was that these commands control spring stiffness (the slope of the line) and have no effect on the threshold of the stretch reflex (the intercept along the abscissa is held fixed). The force-length curves of the antagonists (dashed lines) are represented in the same way (note that agonist stretch unloads the antagonist). Under the assumption that the external load force is zero, model limb position is determined by the intersection of an appropriate pair of solid and dashed curves, a point that defines an equilibrium between equal and opposite forces. The simulated movements in *B* and *D* represent transitions from one equilibrium point to another produced by step changes in agonist stiffness.

The foregoing discussion clearly indicates that movements could be produced by changes in stiffness; however, this does not mean that movements are ordinarily produced in this manner. In fact, the evidence reviewed in *Actions of Control... Motor Servo*, p. 286, *Torque-Angle Relations*, p. 292, and *Equilibrium Point Control*, p. 311 is against the basic postulate that central motor commands set the slope of a force-length relation as assumed in Figure 39C. Instead, it appears that motor commands produce appreciable shifts of the intercept along the abscissa as shown in Figures 25 and 31. In spite of this deficiency, the stiffness control model may prove useful in understanding movement control after dorsal rhizotomy. The latter procedure disables the stretch reflex, in which case central motor commands would be expected to recruit select numbers of units from the motor pool, rather than to set reflex threshold. Correspondingly one would predict that the family of dashed curves in Figure 24 (length-tension curves at different levels of recruitment) would serve as a substitute for the normal family shown in Figure 25. The family of recruitment curves is more accurately characterized by alterations in stiffness than by changes in threshold. These considerations may help to explain

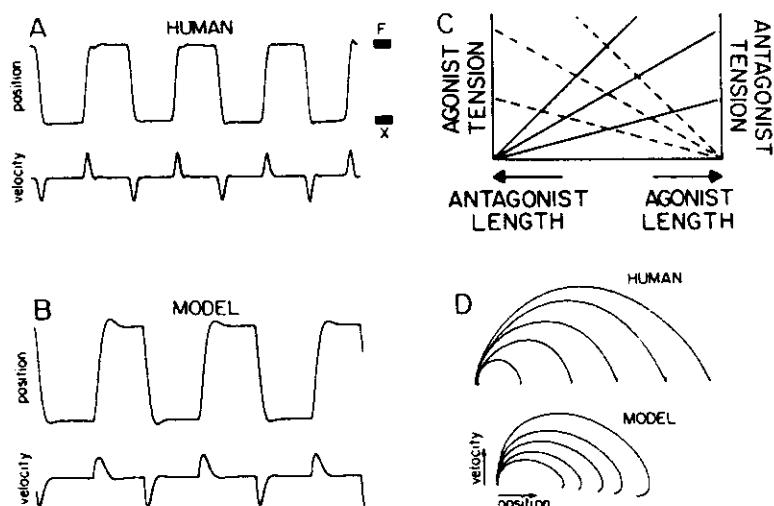


FIG. 39. Movements made by a normal human subject and by an analogue model of the limb. A: position and velocity records of movements made by subject during performance of visually guided step tracking task. B: analogous traces obtained from the model in which movements were produced by a step change in resting spring constant. C: a representation of a model in which the static position of a limb is treated as the point of equilibrium of opposing springs of variable stiffness. D: phase plane plots of movements obtained from human subject and from model. In the model, different movement amplitudes were produced by varying the size of the step in spring constant. [From Cooke (44).]

how deafferented animals are able to maintain reasonable motor control in the absence of stretch reflexes.

#### Perturbations During Movement

In recent years, a number of studies have examined the reflex effects of perturbations interjected in the course of ongoing movement. Such studies, which have focused largely on short-term variations in EMG response, are important in assessing possible interactions between motor servo regulation and descending commands. For example, it is conceivable that motor servo action might be augmented or reduced according to the needs of the situation, which amounts to adaptive gain control (*Adaptive Models*, p. 269). Another possibility is that reflexes would be uniformly suppressed during movement (223), or finally, descending signals and motor servo responses might simply sum in an algebraic fashion. The perturbations are here being used as probes to investigate reflex responses, but the findings are clearly relevant to life situations in which movement is frequently influenced by external environmental constraints.

The EMG response to transient perturbations during motion has been examined in human (54, 55, 91-93, 95, 165, 186, 187), monkey (25, 42), and cat preparations (244). Studies have been performed in a variety of different muscles, performing many different tasks, including visuomotor tracking (186, 187), ballistic movements (50), locomotion (53, 244), and falling (51). A number of similarities to earlier findings in stationary muscles have emerged, as well as a number of apparent differences.

The similarities are that abrupt muscle stretch in-

duces early EMG responses, with time course and latencies comparable to those seen while stationary. In the long thumb flexors, for example, the response to abrupt interruption of voluntary flexion of the thumb may arise as early as 24 ms, a latency comparable to that seen in stationary muscle (186). Comparable medium-latency response components are also evident in shortening thumb muscles, arising at 40-55 ms after perturbation onset (186). Finally, there are also response components that may begin at medium latency but are clearly instruction dependent and appear to represent a form of reaction-time movement that can be released readily by some appropriate stimulus [94, 255]; see *Effect of Instructional Set*, p. 294].

Although the existence of these EMG responses is strong evidence that motor servo action persists in the course of movement, relatively few attempts have been made to compare directly the magnitude of the EMG responses elicited in stationary and moving limbs. One recent study (94) examined reflex transmission during movement by inserting 50-ms torque pulses prior to and early in the course of rapid plantar flexion movements of the human foot. They observed that the short-latency response to perturbation was prominent over the first 100 ms of movement but then went through a period of marked depression. This depression did not necessarily coincide with any period of EMG silence, and it was present at several different stretch velocities and amplitudes. A similar depression has been noted in the H-reflex and tendon jerk responses elicited during phasic voluntary contraction (91, 92). In contrast, Marsden and colleagues (186, 187) provide no evidence of relative inhibition when per-

turbations are introduced during active shortening of the long thumb flexor, although their perturbations were introduced at constant latency, well after the onset of motion, during movements performed at rather slow velocity.

In spite of the differences in EMG response, which could be dependent on the particular muscle used as well as the experimental approach, it appears that differences in EMG response to perturbation do exist in moving muscles; however, it has not yet been shown that these EMG differences result from alterations in CNS transmission or that they carry functional significance. Specifically the EMG changes simply could reflect changes in afferent input from moving limbs (see below), which would make assessment of central gain difficult. Furthermore these EMG changes may not automatically translate into comparable differences in force output, especially in light of possible nonlinearities in the dynamic EMG to force transformation in different muscles.

To pursue this problem of assessment of EMG change further, it is required that any attempt to dissect component contributions to the reflex response include appropriate estimates of the behavior of the controlled system under comparable stimulus conditions. For intact limbs, these estimates would include such things as contributions of inertial, viscous, and elastic properties of skeleton, ligaments, and muscle, excluding any reflex contributions. The only available estimate of this type in intact preparations is that of Bizzi and colleagues (25), who calculated the contributions of reflex action arising during perturbations interrupting voluntary head movement in monkeys. These authors concluded that the time course of positional change of the head was determined largely by the mechanical properties of head and neck musculoskeletal elements. Reflex action was estimated to contribute only 30% to the total force output, an increase deemed insufficient to provide effective load compensation. There was no attempt made to compare the magnitude of reflex action during movement with that observed in stationary muscles.

A number of important additional considerations are relevant to the assessment of reflex responses in moving muscle. First, regarding afferent input, the response of spindle afferents could be quite different for perturbations induced in the course of motion versus those recorded under stationary conditions. The evidence regarding changes in spindle afferent response during motion is so far incomplete; however, our recent studies derived from innervated spindles in the decerebrate cat preparation have shown that responsiveness is depressed during motion. A related issue is that afferent discharge rate during muscle shortening is determined jointly by shortening velocity and by static fusimotor bias (244). Even strong fusimotor activity may not maintain discharge when velocity of shortening exceeds 0.2 rest lengths per second (244). For lower velocities the effect of static fusimotor

action becomes quite prominent. Perturbations introduced in shortening muscles may invoke little or no spindle afferent response unless velocity is less than some critical value, even in the presence of substantial fusimotor activity. Differences in afferent response may also reflect the dependence of a model reference system on load, a point addressed in an earlier section.

A final important aspect of motor servo response is that muscle properties will undoubtedly be strongly influenced by the parameters of the ongoing motion. For example, stiffness and viscosity of muscle are both changed during ongoing movement, which may influence the amplitude of response to a given load.

#### *Compliance, Load Compensation, and Biological Design*

Load compensation, length regulation, and high stiffness are essentially equivalent ways of describing what was once believed to be an important function of the motor servo. However, evidence reviewed at many points in this chapter indicates that motor servo action is not particularly effective in compensating for changes in load. The data further indicate that the stiffness of the stretch reflex is not especially high—it is not much different than the stiffness that could be achieved solely on the basis of muscle mechanical properties (assuming a fully recruited muscle). Two important functional questions that arise from these results are 1) what possible advantages are there to regulating stiffness at a modest level? and 2) how do we achieve load compensation when we need it?

Trajectory a in Figure 30 reviews the springlike response of the motor servo to an increase in load force. In attempting to understand potential advantages, it is helpful to emphasize the compliance of the response rather than its stiffness. Compliance is defined by the ratio of length change to force change, i.e., the inverse of stiffness and the inverse of the slope of trajectory a. While the compliance of the motor servo clearly interferes with load compensation (there is an appreciable increase in length in response to the increase in force), it may be quite beneficial in providing a suitable mechanical interface between the body and its mechanical environment (48, 127). A compliant interface would absorb the impacts of abrupt changes in load, thus attenuating their transmission to the body and head. For example, in the standing posture with knees bent, the regulation of a modest level of stiffness probably insures a good suspension system for the body. Compliance may also be advantageous for the arm musculature. If the arm should encounter an immovable obstacle in the course of a movement, it is better that the musculature yield than continue to control the position of the joint in an unchanging manner, since the latter action would require that the position of the remainder of the body change drastically in order to accommodate the commanded trajectories of joint rotations.

While compliance seems beneficial in some instances, it clearly is not when the task is to make a precise movement or to hold accurately a given limb position. The results reviewed in *Effect of Instructional Set*, p. 294, and *Gain Variation vs. Gain Control*, p. 296, suggest that rigid control of limb position is achieved mainly by the production of reaction-time movements, rather than by increasing the stiffness of the motor servo through a gain-control mechanism. Trajectory a-b in Figure 30 provides a simple hypothetical example of a load-compensated response, presumed to be mediated by an S-R processor (Fig. 13) that emits a new motor command in response to the perturbation. The manifestation of the motor command is a leftward shift in reflex threshold (dashed curve in Fig. 30). The particular response illustrated is a sequential one which characterizes the C traces in Figure 33B; the initial component is a compliant deflection mediated by the motor servo (trajectory a) followed by the response to the new motor command (trajectory b). A clean two-component sequence is not always seen (cf. Fig. 33A), due apparently to the generation of a load-compensating command prior to the completion of the motor servo response. The latter would result in a short-cut trajectory rather than the a-b sequence shown in Figure 30, although the final postural state would be no different. In the example given, load compensation is essentially perfect, since there is no net increase in muscle length even though the greater load persists.

At first thought it seems surprising that the CNS does not utilize the mechanism of adaptive gain control instead of, or in addition to, adaptive S-R processing. One likely disadvantage of the former is that the extent to which length-feedback gain can be increased is quite limited, due to myotatic time delays that promote instability and oscillation. This limitation is discussed in detail in the chapter by Rack in this *Handbook*. Another potential disadvantage is that the use of parametric gain control might unduly complicate the computation of appropriate movement commands, since the latter would have to include corrections based on the current values of stiffness. The theoretical work of Inbar (145) appears to favor the simplicity of signal adaptation (analogous to the selection of appropriate motor commands by an S-R processor) over parametric adaptation. One can also argue on the basis of evolutionary simplicity—organisms clearly require S-R processors, whereas they can get along without parametric gain control, particularly if fast reaction-time processes are available.

Finally, it is interesting to ask why there is any need for stiffness regulation, since the regulated value is not greatly different from muscle mechanical stiffness and since stiffness is not subject to any major adaptive modification. Here it is important to recall that since muscle properties are highly nonlinear, mechanical stiffness varies markedly depending on the initial conditions and on the parameters of length change. Ni-

chols and Houk (226) argued that the improvement in linearity (equivalent to a greater constancy of stiffness) in the presence of reflex action is an important function of the motor servo. The complexity of central motor control would be increased if it were necessary to build corrective components that compensate for muscle nonlinearity into postural and movement commands.

### Summary

Motor commands are distributed to  $\alpha$ -,  $\beta$ -, and  $\gamma$ -motoneurons in combination. Most of the data suggest stereotypic patterns of activation, although the specific pattern may not be quite the same in human versus animal subjects.  $\beta$ -Innervation is more widespread than was formerly believed and is probably important in producing the parallel increases in spindle receptor discharge and force observed under isometric conditions. Data relevant to model reference theories of movement control is judged to be inconclusive at the present time. Regardless of the  $\alpha$ -,  $\beta$ -, and  $\gamma$ -motoneuron makeup of movement commands, the basic outcome seems to be a simple shift in the threshold of the stretch reflex, and considerable progress in understanding movement control has been achieved on this basis.

Movements have been successfully analyzed as transitions from one postural state to another. A postural state is characterized as an equilibrium between stretch reflexes in a given musculature and the forces resulting from mechanical properties of load. Equilibrium is achieved when the muscles are sufficiently stretched to produce forces that exactly counterbalance the load. Movements are produced when a central motor command shifts the reflex thresholds of the musculature, creating a temporary disequilibrium that causes a transition to a new equilibrium state. Perturbations applied in the course of movement alter the time course of motion and provoke reflex responses, many components of which are analogous to those evoked in stationary limbs. Provided reaction-time responses are suppressed, however, the perturbations either do not alter the final postural state, or alter it in a manner predictable from the springlike properties of the motor servo. Thus the motor servo is seen as a final common processor of central motor commands.

One advantage of stiffness regulation is that it provides a springlike interface between the body and its mechanical environment. The compliance of this interface absorbs the impacts of abrupt changes in load, thus attenuating their transmission to the body and head. A disadvantage is that it provides poor load compensation and poor length regulation. Nevertheless, effective load compensation can be achieved by the superposition of appropriate reaction-time movements upon the compliant responses of the motor servo.

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