Regulatory Actions of Human Stretch Reflex

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SUMMARY AND CONCLUSIONS

1. The stretch reflex in the elbow flexor musculature was studied in 23 human subjects. The subjects were required to establish an initial force equivalent to 10% maximum at a prescribed initial length; mechanical disturbances delivered at random times increased load force to 15% or reduced it to 5%. We measured arm force, displacement, and EMG (usually biceps); acceleration was calculated from displacement, and average responses from sets of 10 like trials.

2. Modification of the stretch reflex was studied by comparing average responses obtained with different instructions, but with the same disturbance. The usual introductions were "compensate for arm deflection" and "do not intervene voluntarily." The initial response did not depend on instruction; changes in response that depended on instruction began abruptly after a latent-period which ranged from 70 to 320 ms (measured from force and acceleration), depending on conditions and subject. The latency became longer (10-50 ms) and more variable when the subject did not know the direction of disturbance in advance. This and other observations indicate that modifications of the stretch reflex are not produced by servo actions. They are produced by triggered reactions, which occur at both short and long latencies and which have properties resembling the movements produced in a reaction-time task.

3. We confirmed that most subjects can suppress triggered reactions when the instruction calls for no intervention, leaving an unmodified reflex response. This response consists of a compliant deflection of the arm in the direction of the disturbance.

4. The compensatory actions associated with unmodified stretch (and unloading) reflexes were assessed from EMG responses of biceps. During a 300-ms transient phase, EMG changes were notably asymmetric when responses to symmetric disturbances were compared. Increased force stretched biceps and produced a prominent increase in EMG, whereas decreased force allowed biceps to shorten and produced either an EMG decrease of smaller magnitude or an actual increase. These asymmetric reflex actions produced quite symmetric mechanical responses (arm displacements and forces), which implies the existence of and compensation for nonlinear muscle mechanical properties. This result is discussed in relation to the hypothesis that the function of the stretch reflex is to compensate for variations in muscle properties, thus maintaining stiffness.

5. Effective control of muscle length or joint position does not result from servo action by the stretch reflex. Errors in position are corrected only when triggered reactions are superimposed on the reflex response.

INTRODUCTION

The function of the stretch reflex in man is not well understood. It is usually assumed that reflex action should compensate for changes in the external mechanical load (cf. ref. 25, 36). This assumption is based on the hypothesis that muscle length, by way of feedback from spindle receptors, is the regulated property of the stretch reflex; a rigid regulation of length would prevent load changes from affecting movements or postures (27). However, several authors studying human subjects and other primates (1, 10, 14, 24, 26, 29, 32) have reported that length is not well regulated and that load compensation is poor, unless long latency responses that are presumed to reflect supraspinal mechanisms are superimposed on the segmental stretch reflex. It has been suggested that these long-latency responses are mediated by a pathway from spindle afferents to the motor cortex, either by way of the cerebellum or more directly (10, 28, 31, 35, 37). Phillips (35) proposed that this pathway might constitute the afferent limb of a transcortical servo loop, an extension of the segmental stretch reflex, and other authors have supported this suggestion (10, 24, 30, 37, 41). However, this does not fit with the results obtained by Vallbo (39) who assessed the overall efficacy of muscle-length regulation from the relationship between spindle discharge rate and the force of voluntary contraction in man. A calculation based on quite conservative assumptions indi-
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The present experiments were performed on the elbow flexor musculature in human subjects. In this system the long-latency response that has been attributed to a supraspinal mechanism has a minimal latency of 70 ms in force recordings (16). This is also the response component which is modified when the instructions are changed from "resist" to "let go" (16). The shortest reaction times for force development in response to taps on the limb were 90 ms in Hammond's (16) study, and the mean latency was in excess of 100 ms.

Our first objective was to determine whether modification of the "long-latency" response results from an actual change in reflex responsiveness or, alternatively, from the appearance of superimposed movements. Our results support the latter interpretation and since these delayed movements are events of abrupt onset, we will refer to them as triggered reactions. A second objective was to test the suggestion (4) that the instruction "do not intervene" reveals a stereotypic reflex mechanism. With some reservation we have been able to confirm this, and we will reserve the term "stretch reflex" to refer to this response. A third and major objective was to test the idea that the function of the stretch reflex is to compensate for variations in muscle properties. Our results provide evidence which favors this hypothesis. A fourth objective was to determine the extent to which human subjects are able to control the stiffness of the stretch reflex. In confirmation of work already cited (4), we found no changes in stiffness that were not accounted for by the mechanical effects of cocontraction or by superimposed movements.

METHODS

We used the apparatus shown in Fig. 1 to study responses to mechanical disturbances that were applied to the arms of 23 adult male and female subjects. The subjects held a light-weight (250 g) handle which had strain gauges installed for measuring tension in the cable. The cable attached by way of a pulley (radius, 1.9 cm) to the shaft of a torque motor (Inland T-4036-B). The current delivered to the motor was controlled to produce an initial tension in the cable of 30–50 N, which represented about 10% of the maximal force that the subjects were capable of resisting. The two directions of mechanical disturbance were produced by equal but opposite changes in motor current, which amounted to 50–70% of initial current in different subjects; thus, the arm was never completely unloaded.

The establishment of an initial force was important for two reasons. First, it provided a means of control over the initial excitability of motoneuronal pools, which seemed necessary to...
distinguish controlled changes in reflex responsiveness from other phenomena. Second, it furnished a moderate level of tonic reflex activity that was suitable for studying and comparing responses to both loading and unloading.

Our mechanical arrangement facilitated testing of the elbow flexor musculature in a comfortable and natural situation. The shoulder was in contact with a brace which minimized body movement. Subjects were asked to keep the handle in line between the shoulder and the pulley and to use their wrist like a hinge. This arrangement required the elbow flexor muscles to support the full load produced by the motor, and forearm supination favored biceps activation (5). Shoulder muscles were active in opposition to gravity and in joint stabilization, but could not relieve the load supported by elbow flexors. (The resultant of shoulder moment was a force perpendicular to the cable.)

The load mimicked both inertial and gravitational components of natural loads. The gravitational component was a force proportional to motor current; we will refer to this controlled variable as load force to distinguish it from arm force, which was monitored as cable tension. (The mass of the handle was small in comparison with the equivalent mass of the arm, which was about 1 kg.) The inertial component of external load, derived from the moment of inertia of the motor, was equivalent to a mass of 2.8 kg. If a load having this mass were lifted vertically, the gravitational force would be 27 N, which is approximately the initial force in our experiments. The presence of inertia prevented excessive accelerations and velocities, which tend to synchronize the EMG into peaks and troughs (cf. Fig. 9 of ref 33). Peak acceleration was about 6 m/s² and peak velocity was about 0.2 m/s.

The EMG activity of the biceps muscle was measured by fastening two silver discs (8 mm in diameter) separated by approximately 3 cm over the belly of the muscle, with an indifferent electrode placed laterally. In some cases we also recorded from brachioradialis or triceps. A specially designed preamplifier was taped to the arm in a position which avoided movement artifacts. EMG processing before sampling included rectification and filtering (100 Hz cutoff) to prevent aliasing.

The experiment was conducted under computer control (PDP 11/20). A ready light indicated to the subject when it was time to begin a trial; other lights defined an initial position of the arm which the subject achieved by either flexing or extending the elbow. The angle of the elbow was approximately 90° at the required initial position. The linear displacement of the arm toward and away from the shoulder was proportional to the rotation of the motor shaft, which was measured with a potentiometer. The disturbance was delivered after the arm had remained within a small range (±0.16 cm) of the required initial position for a random time (1–6 s). Sets of 20 trials were conducted in rapid succession. A rest period was allowed between sets and subjects were allowed to rest during a given set if they became fatigued, which seldom occurred.

Records of load force, arm force, displacement, and EMG were sampled at 3- or 5-ms intervals with 12-bit resolution. Ensemble averages and standard deviations were calculated after sorting into sets of like trials, typically 10. Average EMG records were subsequently smoothed with a digital low-pass filter; the 30-ms time constant was chosen to reflect the low-pass characteristics of muscle (15, 16, 34). Acceleration traces were calculated by double numerical differentiation of displacement records. Prior to this operation, displacement samples were scaled up by 16 and filtered with a 3-point sliding window to reduce quantization noise.
RESULTS

We have compared the responses to load change obtained with several different instructions on different occasions, but the two which we used most frequently were "compensate for the deflection of your arm" and "do not intervene voluntarily to compensate for arm deflection." In the first two sections which follow, we describe differences in the responses that depend on prior instructions and make no assumptions concerning whether unmodified stretch reflexes are part of these complex responses. Following this, we present evidence that the responses obtained after the request that the subjects make no intervention can, in selected cases, be regarded as unmodified stretch reflexes.

Responses to disturbances of known direction

Increases in load force that were delivered at random times deflected the arm away from the body (Figs. 1 and 2). The subjects had been instructed either to not intervene or to compensate; a sequence of trials with one instruction was followed by a sequence with the other. When the request was for no intervention, the maintained load force resulted in a maintained displacement of the arm in the direction of the disturbance. When the instruction was to compensate, the time course of displacement was more complex and the final value was often in a direction opposite to the direction of the disturbance; i.e., there was a maintained overcompensation, as illustrated in Fig. 2. This aspect of response cannot be attributed to servo action, since steady-state overcorrection is incompatible with stable operation, whereas the arm was quite stable.

The ensemble averages of the responses obtained with the two instructions were superimposed in order to measure the latency at which differences in responses occurred. These instruction-dependent differences had abrupt onsets, which we will refer to as "departures." For the example shown in Fig. 2, the departures in acceleration, in arm force, and in doubly filtered EMG all had latencies of approximately 70 ms, which suggest that they are analogous to the modifications in stretch response studied by Hammond (16) using a different pair of instructions. Departures in singly filtered EMG occurred at a latency of 60 ms. The unfiltered EMG was unavailable in this instance, but in other instances when it was available we found it too variable to allow reliable estimates of the latencies of departures. The very earliest changes in singly filtered EMG began at 25 ms and did not differ for the two instructions.

The latencies at which departures in acceleration occurred ranged from 70-165 ms in a group of eight subjects tested with the same paradigm. These subjects had latencies that ranged from 80 to 175 ms when the mechanical disturbance was a decrease in load rather than an increase. For half of these subjects the latency was longer with load decreases; in the others, latency did not depend on the direction of the disturbance.

FIG. 2. Dependence of responses to increased load force on instruction. Heavy traces show averaged responses \((n = 10)\) with the instruction "do not intervene" and light traces show averaged responses \((n = 10)\) to the same disturbance with the instruction "compensate." For these records, the direction of disturbance was known in advance; when the direction was unknown (not shown), the latency at which an instruction-dependent difference (departure) in arm force occurred increased from 70 to 80 ms. The EMG was doubly filtered in A and singly filtered in B (30 ms time constant); acceleration was also filtered (15 ms time constant). Calibrations represent 10 N for load force and arm force, 2 cm for arm displacement, and 5 m/s² for acceleration.
Responses to disturbances of unknown direction

The responses described in the previous section were obtained with the subject fully aware of the direction of the impending disturbance. Hence, he always knew the direction of an appropriate response in advance. In the same subjects we studied the responses obtained when the direction of the disturbance was made random, which meant also that the direction of an appropriate response was not known in advance. This difference would not affect performance in a servo system; but, if the corrections were analogous to responses in a reaction-time task, one would expect the latencies to increase and become more variable due to the added element of choice (17, 23, 40). We found a definite increase in latency, and responses became more variable.

The latency at which a departure in acceleration occurred increased by 10-50 ms in 11 out of 16 comparisons involving the 8 subjects already mentioned; in 4 comparisons, the increase was larger and in one there was no change. In a group of 21 subjects, the latency of acceleration departures with unknown disturbances ranged from 80 to 320 ms. The longest ones occurred in subjects who were told that a correct compensatory response was more important than a fast one; the shortest latencies were found when speed was stressed. However, there was considerable overlap between the two groups. It also appeared that latencies were shorter when larger disturbances were used, but this possibility requires further study.

Plots of standard deviation versus time calculated from sets of displacement records indicated that variability of displacement was low (less than 10%) with either instruction during the latent period prior to the departure. An increase in variability (as much as threefold) which began at the latency of the departure was often observed with the compensate instruction, and usually not with the request for no intervention. Variability was more prominent with naive subjects than after a period of practice.

The reasons for the late increase in variability became evident when individual responses were superimposed for comparison. Most of the responses obtained with the request for no intervention (NI) superimposed well on each other, as illustrated for two subjects in Fig. 3. Responses obtained with the request for compensation (C) also superimposed at early times, prior to the latencies at which they differed from the NI responses. A major cause of the variability at later times was a trial-to-trial difference in the timing of a delayed component of response, and it was clear that this component was responsible for the departures described earlier. The delayed component also showed some variation in time course and amplitude and, in occasional trials, it was initially in a direction opposite to the one which resulted in compensation (trace I in Fig. 3B). These inappropriate responses suggest an error in a decision-making process and cannot be explained as the response of a servo system.

Identification of unmodified stretch reflexes

We concluded from the results already presented that the observed modifications of the stretch reflex, regardless of their latencies, were the result of triggered reactions, events which represent selected preprogrammed movements. Thus, one basis for the identification of unmodified stretch reflexes was the absence of triggered reactions. The latter were usually easy to recognize in individual displacement traces (cf. Fig. 3), although when they were small in amplitude and occurred during the initial deflection of the arm they were sometimes difficult to
detect. Another basis for identifying the unmodified response was a low variability which continued throughout the period of observation. The sensitivity of this test was improved by restricting our analysis to cases in which the direction of the disturbance was random since the modifications, when present, were more variable in this circumstance. Randomization of direction and distraction of the subject were both effective in delaying or inhibiting triggered reactions. Delaying them helped in their identification since triggered reactions were more easily distinguished when they occurred on the plateau of the stretch reflex (Fig. 3B) than when they occurred during the 100- to 200-ms transient phase (Fig. 3A).

By using these various criteria and procedures, we were able to substantiate the assumption that responses which are relatively free of modifications, when present, were more variable in this circumstance. Randomization of direction and distraction of the subject were both effective in delaying or inhibiting triggered reactions. Delaying them helped in their identification since triggered reactions were more easily distinguished when they occurred on the plateau of the stretch reflex (Fig. 3B) than when they occurred during the 100- to 200-ms transient phase (Fig. 3A).

**EMG changes accompanying reflex actions**

The earliest changes in EMG activity occurred at latencies ranging from 20 to 65 ms in a group of 21 subjects. These latencies preceded those for departures in EMG for all subjects although the latency of the latter was shorter or nearly the same for some subjects as the latency of the former was for others. For each subject it was clear that the first change in EMG represented the onset of reflex action, and this change did not depend on whether the direction of disturbance was random or known.

The results obtained after the instruction not to intervene indicated that reflex action, once initiated, continued for the duration of the record. This finding suggests that the long-latency component of stretch response described in the introduction is, in general, a composite response made up of a reflex action together with a triggered reaction. When we compared the extra increment in EMG attributable to a triggered reaction with the increment attributable to reflex action, the two were sometimes of comparable magnitude (compare heavy traces with the difference between heavy and light traces in Fig. 2).

The remainder of this section deals exclusively with EMG responses that were attributed to reflex action.

**REFLEX RESPONSES TO INCREASED LOAD FORCE.** None of our subjects showed prominent EMG peaks at short latencies in response to increased load force. Instead, there was a broad elevation reaching a maximum at 100–200 ms (Fig. 4, heavy traces). This indicated that the monosynaptic pathway was not activated appreciably at its minimal latency, but did not exclude a participation in the later response along with polysynaptic pathways. The absence of a sharp, monosynaptic peak was attributed to the modest accelerations and stretch velocities achieved; as mentioned earlier, the abruptness of the disturbance was deliberately limited so as to prevent the EMG from becoming synchronized.

The responses of most subjects showed some cyclical behavior before settling to a steady value, and there were correlated changes in force. In the example shown in Fig. 4A cyclic variations are prominent, whereas in Fig. 4B they are less noticeable. In none of our subjects did the periods of reduced activity represent actual silent periods, as confirmed by examining the records from individual trials.

**REFLEX RESPONSES TO DECREASED LOAD FORCE.** Typical EMG responses to decreased load force are shown by the light traces in Fig. 4. There was an initial, brief period of decreased activity (sometimes absent, as in Fig. 4B), followed by a longer period during which activity increased, frequently to values above the initial, predisturbance level; subsequently, the EMG decreased to a value below the initial level. The phase of increase always began while the biceps continued to shorten. Previous studies of the EMG responses to unloading have usually involved a complete, or nearly complete, removal of the initial force on the limb. Under these circumstances, the first EMG change is often a silent period that may be ended by a "terminal motor volley" (2) or by a more complex pattern (38). Under our conditions of partial unloading, we rarely observed a period of EMG silence of sufficient length to be termed a silent period, and the terminal volley was replaced by the broad increase in EMG activity described.

The detailed features of the EMG responses differed between individuals even though the initial mechanical conditions and the load change were approximately the same (compare Fig. 4A and B). However, when a single subject was examined on different days, the responses were quite similar. This suggests that the differences between subjects are genuine and are not due to differences that might be produced by the different placements of the electrodes.

**ASYMMETRY OF EMG RESPONSES TO SYMMETRICAL LOAD CHANGES.** The EMG responses to symmetrical changes in load were notably
asymmetric in all of our subjects. The EMG always increased when the elbow flexors were stretched by loading; but, when these muscles were allowed to shorten by unloading, there was either a decrease of smaller magnitude, or an actual increase. In the extreme case the asymmetry was so marked that the responses to unloading looked very much like the responses to loading (Fig. 4B); in other cases the difference was less marked (Fig. 4A). We consider this asymmetry to be one of the characteristic features of the self-regulating pathways to and from individual muscles, a matter we will return to in the discussion. However, other possible interpretations required consideration.

The first possibility was that the action of other muscles at the elbow joint complemented the asymmetry of the biceps EMG. We found, however, that the brachioradialis EMG either was similar in time course to that of biceps or did not change appreciably, and that the triceps EMG was undetectable during reflex responses in either direction. The initial flexor tonus, the limited amount of unloading, and the slowing of the movement by the inertial component of the load were probably responsible for the absence of triceps activity. (The triceps was active in some subjects in association with triggered movements, and in other cases that will be considered later.)

A second possibility was that EMG silence during unloading contributed to the asymmetry. However, silent periods were seen in only a few subjects; when present they lasted less than 20 ms and appeared only when the initial force was adjusted to a low level.

The third possibility was that the relationship between EMG activity and contractile force was curvilinear, as reported to be the case for some subjects by Bouisset (6). However, monotonic curvilinearity cannot explain an increase in EMG above the initial value during shortening. Furthermore, the data in Fig. 4 are from two subjects with linear EMG-force relations.1 It

### Figure 4

Comparison of symmetry of arm force, displacement, and EMG responses (means in all cases) to symmetric changes in load force for two subjects. The heavy traces represent responses to step increases in load force and the light traces represent responses to equal but opposite decreases. The EMG responses were singly filtered (30 ms time constant) and were calibrated in units of force. Both subjects (but especially B) showed force and displacement responses which were much more symmetric than were the EMG responses. Note, however, that the steady-state changes in EMG were more symmetric.

1 The EMG was calibrated by asking the subject to hold his arm at the same initial position with different steady loads. The steady-state relation between EMG and force at constant length was used to provide offset and multiplicative (scale) constants.
may be concluded that curvilinearity does not account for the asymmetry, although it may contribute to it in some subjects.

**Attempts to alter stiffness of stretch reflex**

We considered that the gain of the stretch reflex might be subject to presetting, such that the stiffness of the limb might be altered (INTRODUCTION). In six subjects we tried various instructions (e.g., "be rigid," "resist," "minimize arm deflection," "cocontract"), but found that none resulted in any appreciable modification of response at times prior to the occurrence of a triggered movement except when the subject succeeded in cocontracting the elbow antagonists. We judged when cocontraction occurred either by direct recording from triceps or by observing an increase in the EMG of biceps when the initial force was unchanged. While one can readily cocontract when a joint is not initially loaded, it is more difficult to do this when the limb is preloaded, as it was in our experiments. Cocontraction doubled the stiffness in some cases, but the change was attributable to purely mechanical effects since we noted no appreciable alterations in the EMG responses other than the upward shift of the initial level. In the usual cases in which there was no apparent cocontraction, subjects accomplished the requested rigidity by producing a triggered reaction. We concluded that the responsiveness of the stretch reflex was not appreciably modified as a result of these different instructions to our subjects.

**DISCUSSION**

Our results provide qualitative support for the hypothesis that the function of the stretch reflex is to compensate for variations in muscle properties, rather than for changes in load, and that the regulated property may be stiffness (20, 33). This interpretation is based on knowledge of the manner in which mechanical properties of muscle depend on the direction of length change.

If a muscle activated by electrical stimulation is suddenly released from an isometric length, the force falls precipitously in a manner that is predicted by the force-velocity relationship (18). (The velocity dependence of force during shortening is probably much more important than the length dependence at the velocities prevalent in our experiments (42).) Thus, the stiffness, calculated as the decrement in force divided by the decrement in length, is high and positive. The velocity dependence during shortening is more complex and cannot be characterized by any single force-velocity curve (22). However, the available data indicate that the initial stiffness of the muscle is high and comparable to that prevalent with release, whereas stiffness decreases markedly and may become negative when the amount of stretch exceeds approximately 1% of the rest length of muscle fibers (22, 33). As a result of these nonlinear features, the mechanical stiffness presented by active muscle fibers should be less during lengthening than during shortening, provided length change exceeds 1%. 2 3

In our experiments, motor units in biceps that were active at the required initial force must have contributed a mechanical stiffness opposing length change, which was then modulated by motor output. The prominent increase in EMG that occurred when the biceps was loaded indicated that the output of the stretch reflex was appropriate to compensate for the expected reduction in muscular stiffness during lengthening. The decrease in EMG that occurred when the biceps was unloaded was smaller in magnitude, which suggested that motor output did not appreciably increase stiffness during shortening, when muscular stiffness would be expected to remain high. In fact, the EMG responses of many subjects actually increased during the transient phase of shortening, suggesting that motor output acted to reduce stiffness. The observed asymmetry of motor output thus supports the hypothesis that the actions of the stretch reflex compensate for variations in muscle stiffness. Compensatory action was quite effective in most subjects, for the displacements of the arm and the changes in arm force that resulted

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2 The change in biceps length was estimated to be 0.14 times the wrist displacement based on the lever ratio given by Wilke (42) and an angle of 45° between the forearm and the direction of displacement. If we assume a typical displacement of 1.5 cm and a biceps muscle fiber length of 10 cm, the change in fiber length in our experiments would have been approximately 2%. For the purpose of this calculation we ignored the smaller changes in biceps length resulting from shoulder rotation.

3 It might be objected that these mechanical properties are special features of the animal muscles that have been studied and that human muscles behave quite differently. While this seemed unlikely, we searched for evidence that the elbow flexors in man presented less mechanical resistance to lengthening than to shortening. Cavagna and collaborators (7) demonstrated that the relationship between work and velocity during maximal voluntary contractions of the elbow flexors is asymmetrical with respect to lengthening and shortening. From their data it can be inferred that, when averaged over the cycle of length change investigated, there was less increase in force during lengthening than there was decrease during shortening.
from equal but opposite changes in load force were approximately symmetric (Fig. 4).4

Recently several authors have favored the hypothesis which was originally put forth by Phillips (35) that in primates and man a transcortical servo loop, or stretch reflex, dominates the segmental stretch reflex (10, 25, 30, 41). Our results bear on this question to the extent that we are able to compare reflex action in human subjects with that observed in decerebrate cats, which lack the postulated "transcortical servo loop." A qualitative comparison indicates that the actions of the stretch reflex are similar in the two cases; both show the rather characteristic form of asymmetry described in the previous section. The major difference between the stretch reflex in humans and in decerebrate cats is that the latter lack the superimposed movements which we refer to as triggered reactions. This obviously suggests, but does not prove, that the demonstrated transcortical pathway might be responsible for conducting or controlling short-latency triggered reactions, rather than mediating servo actions. This is an important distinction since the performance characteristics and neural mechanisms of the two types of system are probably very different.

Our results did suggest one potentially important difference between stretch reflexes in humans and in decerebrate cats. The phase of EMG increase during shortening is a feature that has not been observed in decerebrate preparations (33; unpublished observations). Since tendon organ discharge should be reduced by the decreased force during shortening, a removal of Ib inhibition is probably an important factor contributing to the EMG increase. The absence of this phase in the decerebrate may result from a low gain in the Ib pathway, which is believed to be a characteristic of the decerebrate state (9, 19, 21).

The phase of EMG increase during shortening is of special functional interest since it represents a reflex action assisting length change, rather than opposing it. In contemplating the significance of this result, it is important to consider that a rigid control of muscle length is not required for body stability; the only fundamental requirements are that forces be adjusted to counterbalance external loads and that there be damping. The stretch reflex meets these requirements while providing a springlike property which imposes a compliant mechanical interface between the body and the environment. The importance of compliance may be its ability to absorb the impact of a sudden change in load, thus attenuating transmission of the disturbance to the body and head (20). When a rigid control of position is required, it is available by the production of short-latency triggered reactions.

There are numerous reports indicating that the responsiveness, or gain, of the stretch reflex is modified when the initial conditions are changed (e.g., ref 21, 25), but these modifications may be due to system nonlinearities rather than to a specific neural control of responsiveness. If there are brain mechanisms which act to alter the gain of the stretch reflex, it should be possible to demonstrate them when the initial force and length are controlled, as in our experiments. Our failure to find such changes agrees with the results reported by Asatryan and Fel’dman (4), and suggests that gain change may not be a usual mechanism by which the stretch reflex is controlled.

Our results indicated that triggered reactions cannot be attributed to the actions of a servo system. At present we do not know how to characterize the neural systems which produce triggered reactions, except to indicate that there is probably a sequence of processes beginning with the detection of specific sensory cues and ending with the selection of appropriate responses. The presetting of these processes that results when different instructions are given to the subject must somehow establish different criteria for detection and associate with each an appropriate preprogrammed movement command. Discovery of the neural mechanisms responsible for these actions would probably lead to a better understanding of voluntary movement control in general.

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4 Measured values of the static stiffness of the limb ranged from 5 to 15 N/cm in different subjects and were not appreciably different for stretch and release. Transient properties of stiffness will be dealt with in a separate report.

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