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Adaptive Plasticity in Primate Spinal Stretch Reflex: Initial Development

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SUMMARY AND CONCLUSIONS

1. Description of the neuronal and synaptic bases of memory in the vertebrate central nervous system (CNS) requires a CNS stimulus-response pathway that is defined and accessible, has the capacity for adaptive change, and clearly contains the responsible substrates. This study was an attempt to determine whether the spinal stretch reflex (SSR), the initial, purely spinal, portion of the muscle stretch response, which satisfies the first requirement, also satisfies the second, capacity for adaptive change.

2. Monkeys prepared with chronic fine-wire biceps electromyographic (EMG) electrodes were trained to maintain elbow position and a given level of biceps background EMG activity against constant extension torque. At random times, a brief additional extension torque pulse extended the elbow and elicited the biceps SSR. Under the control mode, reward always followed. Under the SSR↑ or SSR↓ mode, reward followed only if the absolute value of biceps EMG from 14 to 24 ms after stretch onset (the SSR interval) was above or below a set value. Animals performed 3,000–6,000 trials/day over data-collection periods of up to 15 mo.

3. Background EMG and the initial 30 ms of pulse-induced extension remained stable throughout data collection.

4. Under the SSR↑ or SSR↓ mode, SSR amplitude (EMG amplitude in the SSR interval minus background EMG amplitude) changed appropriately. Change was evident in 5–10 days and progressed over at least 4 wk. The SSR increased (SSR↑) to 140–190% control amplitude or decreased (SSR↓) to 22–79%. SSR change did not regress over 12-day gaps in task performance.

5. A second pair of biceps electrodes, monitored simultaneously, supplied comparable data, indicating that SSR amplitude change occurred throughout the muscle.

6. Beyond 40 ms after pulse onset, elbow extension was inversely correlated with SSR amplitude. The delay between the SSR and its apparent effect on movement is consistent with expected motor-unit contraction time.

7. The data demonstrate that the SSR is capable of adaptive change. At present the most likely site(s) of the mechanism of SSR amplitude change are the Ia synapse and/or the muscle spindle.

8. Available related evidence suggests persistent segmental change may in fact come to mediate SSR amplitude change. If so, such segmental change would constitute a technically accessible fragment of a memory.

INTRODUCTION

The central nervous system (CNS) substrates of memory, or long-term adaptive change, in vertebrates remain unknown for two reasons (51). First, the CNS pathways subserving most stimulus-response sequences are intricate, incompletely defined, and not readily accessible, rendering it difficult or impossible to define changes. Second, since neurons in these pathways may be affected by input from other CNS regions, any changes noted might simply be due to tonic input originating elsewhere. Solution of these problems is contingent on defining a CNS stimulus-response pathway that *1*) is

defined and accessible anatomically and physiologically, *2*) is capable of adaptive change, and *3*) unequivocally contains the responsible substrates. The initial, segmentally mediated response to sudden muscle stretch satisfies the first criterion. The present study demonstrates that it satisfies the second and, in conjunction with other data, suggests that it also satisfies the third.

The spinal stretch reflex (SSR), the initial, purely spinal, portion of the muscle stretch response, is also referred to as M1 (31), or the tendon jerk. It is the fastest and simplest stimulus-response sequence of which the CNS is capable. In the monkey biceps, for example, the SSR begins 12–14 ms after muscle stretch onset, peaks at 18–20 ms, and declines by 30 ms, before onset of the later components of the stretch response (12, 55). It is mediated entirely at the segmental level, largely by the two-neuron monosynaptic arc made up of the Ia-afferent fiber from the muscle spindle (or, to a lesser degree, the group II afferent fiber from the spindle (48)), its synapse on the α-motoneuron, and the α-motoneuron itself. Other afferents play a minor role (24, 36).

If initial muscle length and initial, or background, α-motoneuron tone are constant, SSR amplitude is dependent on muscle spindle sensitivity and on Ia synaptic function. Spindle sensitivity is controlled by γ-motoneuron tone (22, 37) and possibly also by sympathetic activity (4, 16, 28, 44). The Ia synapse is subject to presynaptic control originating both peripherally and centrally (3, 9, 34). It is not clear at present to what extent primates can use these or other mechanisms to control SSR amplitude without change in initial muscle length or background EMG.

Since its initial description a century ago (30), neurologists have used the Jendrassik maneuver, in which patients are asked to tonically contract remote muscles, to augment the tendon jerk, the clinical equivalent of the SSR. For example, if an individual clenches his fists, the knee jerk elicited by patellar tap typically increases in amplitude. Studies indicate that this tendon jerk increase is accompanied by comparable increase in the H-reflex, the monosynaptic α-motoneuron response to electrical stimulation of the Ia-afferents, and is not accompanied by change in spindle behavior (7, 10). Thus, it appears that the effect is mediated at the Ia synapse or the α-motoneuron, rather than at the muscle spindle.

Laboratory studies in humans and nonhuman primates, beginning with Hammond (21), also suggest that control over SSR amplitude is possible. In the standard design, subjects maintain a specified limb position (such as elbow angle) against a constant force on which brief additional force pulses are superimposed. Subjects are instructed either to resist the perturbations caused by the pulses or to let go. While instruction has its greatest effects on later components of the stretch response, most studies (17–20, 29), though not all (12, 21, 47), report modest appropriate effects on the SSR. SSR amplitude tends to be greater with the resist instruction and smaller with the let-go instruction. While initial muscle length and background force opposed have been the same for the two instructions, background EMG has generally not been carefully monitored. Thus, as with the Jendrassik maneuver, effects on SSR amplitude might be due to undetected changes in α-motoneuron tone rather than to changes in the reflex arc itself.

The goal of the present study was to determine whether monkeys can control SSR amplitude without change in initial muscle length or background α-motoneuron tone and to define the time course and magnitude of such change. The design, based on the perturbation task, had three special features. First, it required not only a given initial position against a given background force but also a given level of background EMG measured as absolute value (equivalent to rectification). Second, because available evidence (see DISCUSSION) suggested that substantial SSR amplitude change would only occur with prolonged training during which the muscle was essentially totally devoted to the task, the task was available to the animal continuously over days. Third, to further increase the likelihood of SSR change, reward was made directly dependent on SSR amplitude itself, as measured by EMG. Two brief preliminary reports of some of our findings have appeared (52, 54).

METHODS

Animal preparation and environment

Subjects were 11 monkeys (6 *Macaca nemestrina,* 5 *Macaca mulatta,* male, 5–7 kg). Under general

anesthesia, four Teflon-coated 10-stranded (25-μm strand diameter) stainless steel fine-wire EMG electrodes (5) were implanted in one biceps muscle as follows. The skin was incised and the final 1.5 cm of each electrode was stripped and inserted into the tip of a 22-gauge hypodermic needle. Beginning near the midpoint of the muscle, the needle was passed obliquely through the muscle in a slightly wiggling course to promote more secure anchoring. Withdrawal of the needle left the wire in place through the muscle belly in a fishhook form. The four electrodes entered near the midpoint of the muscle and fanned out distally and proximally so that they were spaced along the long axis of the muscle with 1–2 cm between the bare tips. The most distal and proximal electrodes were the primary pair (biceps I) and the middle two were the secondary pair (biceps II). The wires passed subcutaneously to an exit either in the proximal forearm or in the midback (in which case a custom-made cloth jacket protected the exit) and were attached to a small connector plug. Electrodes of this type become firmly anchored in the muscle. They are not susceptible to significant movement artifacts and provide stable recordings indefinitely. We have used given pairs for periods of over a year without detectable change in their performance and have then reoperated and confirmed that they had remained in the same position. Wire breaks, which occur rarely, are immediately evident due to marked 60-Hz noise.

Data were collected from each animal over 1–15 mo. This time was composed of 1- to 12-wk data-collection periods interspersed with 1- to 5-wk rest periods. Animals spent rest periods in individual standard primate cages with the EMG electrode connector plug coated with Silastic and buried under the skin. During data-collection periods, animals remained in the laboratory continuously. This design allowed continuous data collection and ensured that the implanted arm was essentially totally devoted to the task described below. As indicated (INTRODUCTION and DISCUSSION), these factors were thought necessary to the success of the study and results have supported this assumption.

The laboratory accommodated four monkeys at a time, in two 2 m x 1 m frames. Each frame accommodated two monkeys facing each other 1 m apart. Each monkey sat on a 1 m x 1 m smooth, flexible grid restrained by a loosely fitting Plexiglas neck collar. The forearm of the implanted arm rested in a custom-made cast fixed at the elbow to a torque motor shaft (see below). Legs and unimplanted arm were free, and the monkey was able to assume a variety of natural waking and sleeping postures (25). Laboratory lighting, controlled by a timer, was reduced from 2100 to 0600. Monkeys ate standard chow from food dishes replenished 4 times per day and received fresh fruit once per day. Daily fluid intake (composed of earned rewards and daily supplements) satisfied established requirements (15). Each animal received repeated meticulous total body inspections. Occasional incipient abrasions received immediate appropriate treatment. Animals remained active, free of significant abrasions, and in good health throughout study.

Equipment

The design is shown in Fig. 1*A*. Each animal's forearm rested in a custom-made cast fixed at the elbow to the shaft of a torque motor that rotated in the plane of elbow flexion and extension. The arm was abducted 60–70° at the shoulder and upper arm position was fixed by a padded Plexiglas brace. The plane of rotation at the elbow was horizontal. The hand grasped a small vertical post in the end of the cast. Thus arm, forearm, and hand postures were controlled. The torque motor exerted a constant modest (0.5 nm) extension torque that tended to extend the elbow toward a padded stop at 130°. Elbow angle was monitored by a rotary variable differential transformer (RVDT) on the bottom of the motor shaft. EMG from the implanted electrode pairs was differentially amplified (×1,000; bandpass, 10–2,000 Hz).

A DEC 11/34 minicomputer system interfaced with all four monkeys simultaneously and continuously. It digitized elbow angle at 500 Hz and biceps I EMG at a minimum of 1,000 Hz (usually 2,000 Hz). In 5 of the 11 monkeys it simultaneously digitized biceps II EMG. The computer recorded the absolute value of the digitized EMG (equivalent to full-wave rectification). It controlled the four digital outputs visible in Fig. 1*A*: *1*) an upper dim green light that simply indicated that the system was running, *2*) a lower bright white light that indicated that elbow angle was correct (see below), *3*) a brief pulse of additional extension torque from the motor, and *4*) a brief reward squirt from the syringe-solenoid system mounted 0.5 m in front of and above the monkey.

Task

Via its two inputs (EMG and elbow angle) and four digital outputs ("system on" light, "correct elbow angle" light, extension torque pulse, and reward pulse) per monkey, the computer monitored and controlled the task illustrated in Fig. 1*B*. The initial, background part of the task required the monkey *1*) to maintain elbow angle at 90° (±1.5°) against the constant extension torque exerted by the torque motor for a period that varied randomly from 1.2 to 1.8 s and *2*) to maintain the average absolute value of biceps I EMG for the last 200

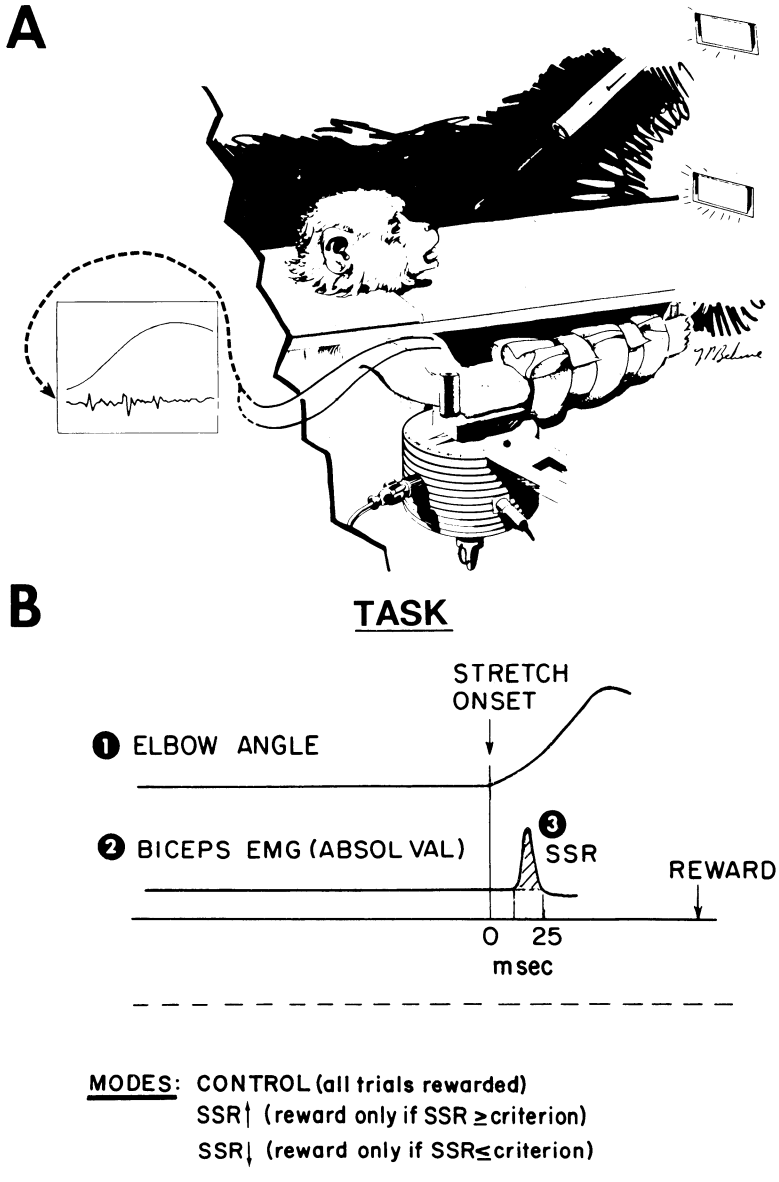

FIG. 1. Experimental design. *A*: animal performing task. Implanted arm rests in a cast attached to torque motor shaft. Elbow angle is monitored by rotary variable differential transformer (RVDT) below motor. Chronically implanted fine-wire electrodes, which exit at midback or upper forearm, monitor EMG. Dim upper light indicates system is on. Bright lower light indicates correct elbow angle. Solenoid-powered syringe delivers reward squirt. *B*: task performed by animal. The animal maintains elbow angle, 1, and biceps EMG, 2, within preset ranges for a randomly varying 1.2- to 1.8-s period against constant background extension torque from the motor. At the end of this period, a brief pulse of additional extension torque extends the elbow and elicits the biceps spinal stretch reflex (SSR). Under the control mode, reward always occurs 200 ms following pulse onset. Under the SSR↑ or SSR↓ modes, reward occurs only if SSR amplitude, 3, is above or below a preset value.

ms of this period within a set range. First, the monkey was trained over 7–10 days to maintain correct elbow angle (indicated by the bright white light). The average absolute value of biceps EMG accompanying this position maintenance was simply monitored. Then EMG limits were imposed so that, from then on, background EMG amplitude for each individual trial was close to this value. If the monkey

fulfilled both parts of the task, the computer immediately delivered a very brief (usually 20 ms) pulse of additional extension torque (about 0.5 nm) via the motor. This pulse transiently extended the elbow. While the electrical time constant of the torque motor was only 3 ms, the inertia of the cast and forearm made the brief extension somewhat slower. Depending on the monkey, rate at 10 ms after pulse onset was 25–30°/s and maximum rate achieved was 60–80°/s. Extension peaked at 80–90 ms and then returned to the initial position over a similar period. For a given monkey, the initial 30 ms of extension was very stable.

This sudden stretch elicited a spinal stretch reflex (SSR) from the biceps. The SSR was evident in the EMG at 12–13 ms, peaked about 18–20 ms, and declined by 30 ms. It was clearly distinct from later components of the stretch response (which, once monkeys were trained, were quite minimal (Fig. 7), probably because the pulse was so brief and no further response was demanded by the task). The computer continued to digitize elbow angle and biceps EMG following pulse onset. The 100 ms following pulse onset was divided into a series of equal windows, usually 50 windows each 2 ms long. The computer measured elbow angle at the end of each window and calculated the average absolute value of biceps EMG within each window. Thus, in the usual case, with 50 windows and the computer digitizing EMG at 2,000 Hz, four values were averaged to determine each window's EMG amplitude. Finally, the computer gave a reward squirt 200 ms after pulse onset.

Thus, in a quite stereotyped manner, the monkey achieved proper elbow angle, held it the required period with proper EMG amplitude, felt the brief pulse, opened its mouth, received the reward squirt, and at nearly the same time again assumed correct elbow angle for the next trial. Monkeys typically completed 10–15 trials/min and worked 5–7 h/day. Thus each performed 3,000–6,000 trials/day. Animals worked most intensively during the day when the laboratory lights were bright (0600–2100), but all did perform substantial numbers of trials during the night when the lights were dim (2100–0600) (55).

As shown in Fig. 1*B*, this task operated under any one of three modes. In the control mode, all trials were rewarded. The monkey simply maintained correct elbow angle and correct biceps I EMG, received the brief extension pulse, and was rewarded. In the SSR↑ mode, reward occurred only if biceps I EMG amplitude in the SSR interval (typically defined as 14–24 ms after pulse onset) was above a criterion value. In the SSR↓ mode, it occurred only if EMG amplitude in this interval was below a criterion value. At the start of the SSR↑ or SSR↓ mode, the criterion value was chosen on the basis of the control period data so as to reward about 50% of the trials. As SSR amplitude gradually changed in subsequent days and weeks and reward percentage consequently increased, the criterion value was periodically changed to reduce the percentage back toward 50%, and thus prompt further SSR change.

Data

The computer provided a data summary for each monkey every 3 h and a grand summary every 24 h (see Fig. 2). Each summary included number of trials, number of rewards, average initial elbow angle and biceps I background EMG amplitude, and average elbow angle and biceps I EMG amplitude for each 2-ms postpulse interval out to 100 ms. It also provided average biceps I SSR amplitude, calculated as average EMG amplitude in the SSR interval minus average background EMG amplitude and presented in units of average background EMG amplitude. Finally, it provided a histogram of SSR interval EMG amplitudes for all the day's trials. Figure 2 shows a typical grand summary. In addition, analog recordings of series of several hundred trials were made periodically on an instrumentation tape recorder. These recordings aided monitoring of system calibration and furnished raw EMG and elbow-angle data from single trials for examination and illustration. We emphasize that all EMG measurements by the computer were absolute value. Such measurement is equivalent to that obtained by full-wave rectification. (It is in fact somewhat superior, since it avoids the slight error introduced by the diodes used in analog rectification.)

For the five monkeys in which a second simultaneous EMG channel was available, biceps II EMG was monitored at least 1 of 4 days (on the other days, the second channel was devoted to synergist and antagonist muscles (56)). Thus, for these animals we have substantial data on biceps II background EMG and SSR amplitude.

Data collection and animal performance

As noted above, data were obtained from each animal for up to 15 mo, with periods of nonperformance interspersed. Since background EMG and SSR amplitude depended on the electrode pair, the same pair, biceps I, was used throughout data collection. Animals always worked initially under the control mode (usually for 10–12 days) and then under the SSR↑ or SSR↓ mode (for up to 100 days). During subsequent months a number of monkeys were exposed to mode reversal (SSR↑ to SSR↓, or vice versa) (53) and/or prolonged non-performance periods, (These data will be covered in a subsequent report.) The present report deals primarily with the control data and the initial development of adaptive SSR amplitude change. All EMG data presented here are from biceps I, the primary electrode pair, except for the section that deals specifically with biceps II results.

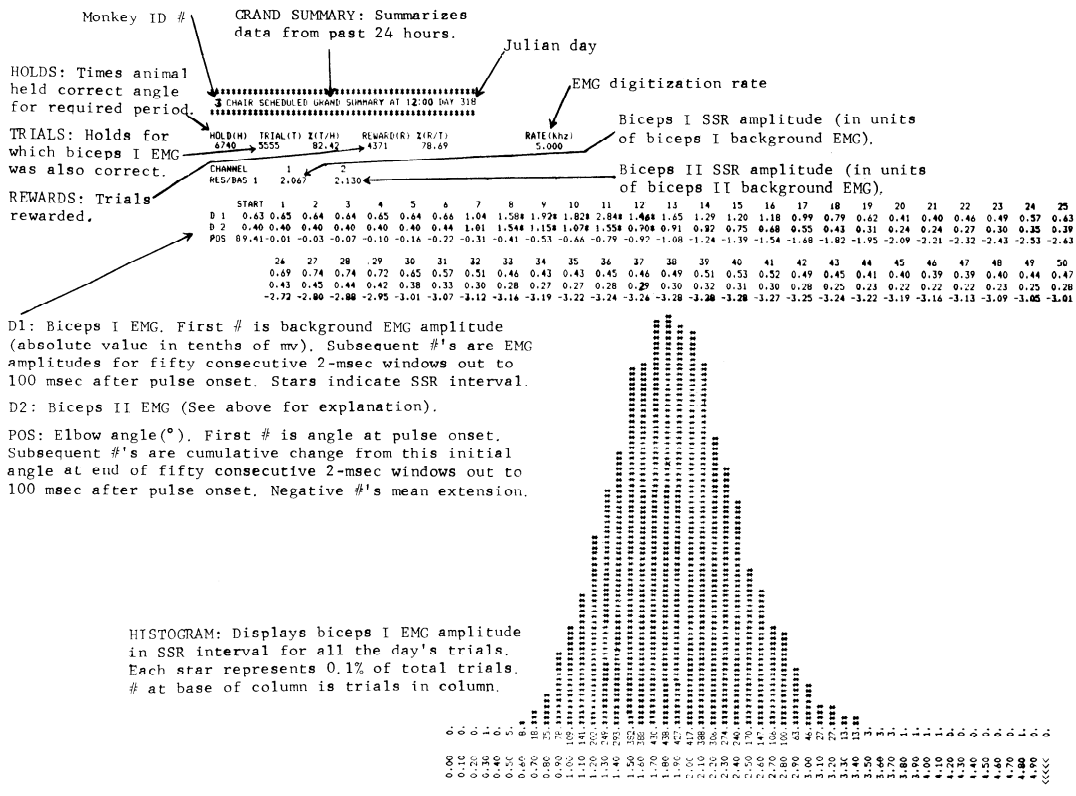

FIG. 2. Sample of grand summary of 24-h data from one monkey. The computer provides such a summary for each animal each day. It also provides comparable summaries every 3 h throughout the day. See text for full explanations of terms.

Throughout data collection from each animal, we noted no significant changes in general posture, arm or hand position, daily performance schedule, or other aspects of animal behavior. An about 3% decrease in arm and forearm circumferences and an about 10-deg decrease at each extreme of elbow movement occurred in the implanted arm in the initial months of data collection. These slight changes were not related to training mode. They were attributed to the continuous commitment of the extremity to the experimental task. As noted above, the initial 30 ms of pulse-induced extension remained stable throughout data collection, further supporting the conclusion that no significant changes in muscle mechanical state took place.

RESULTS

Control data

Biceps I background EMG amplitude was typically 60- to 70-μV absolute value, equivalent to a sine wave of 200-μV peak-to-peak amplitude. This was estimated to be about 10–15% of the muscle's maximum EMG output. Depending on the monkey and the electrode pair, SSR amplitude, that is, EMG amplitude in the SSR interval minus background EMG amplitude, was 1.5–4.5 times background EMG amplitude.

For each monkey, the first 30 ms of pulse-induced stretch, biceps background EMG amplitude, and biceps SSR amplitude remained stable throughout the control period.[1] The standard deviation of daily average values was <3% for the initial 30 ms of extension, <5% for background EMG, and 5–12% for SSR amplitude. Figure 3 shows

[1] In two monkeys, substantial changes in SSR amplitude occurred over the initial weeks of performance. Control data collection did not begin until SSR amplitude had clearly stabilized. These spontaneous changes reduced phasic EMG activity, thereby improving performance economy. Thus they may have represented self-training (see first paragraph of DISCUSSION).

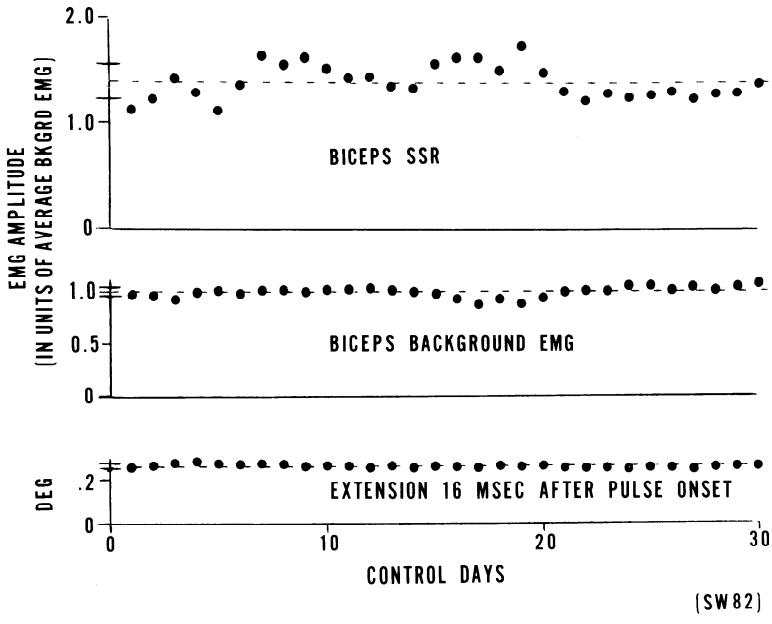

FIG. 3. Daily average extension at 16 ms after pulse onset, background EMG, and SSR amplitude over a 30-day control period in one monkey (4,000 trials/day average). Background EMG and SSR amplitude are in units of average absolute value of background EMG over the entire period. Elbow extension at 16 ms following pulse onset is given in degrees. Standard deviations are indicated. The three measures remain stable throughout the period.

daily averages for extension at 16 ms, biceps background EMG, and biceps SSR amplitude for one monkey during a 30-day control period. The lines show mean values (±1 SD) over the entire period. Similarly stable data were obtained from each monkey prior to onset of the SSR↑ or SSR↓ mode.

SSR↑ mode

Following collection of control data, five monkeys were switched to the SSR↑ mode for a 20- to 80-day period. Background EMG amplitude and the initial 30 ms of pulse-induced extension remained stable. Daily SSR amplitude increased significantly in all five monkeys. Increase became evident in 5–10 days and progressed slowly over subsequent weeks. For the five monkeys, final values were 140, 141, 145, 167, and 190% of control.

Figure 4 shows representative data. Figure 4*A* displays daily SSR amplitudes during the control period and the SSR↑ period for one monkey. SSR increase occurs gradually over weeks. It does not decline over a 12-day home-cage break. Figure 4*B* shows histograms from a monkey before and after SSR↑ onset, each giving the distribution of single-trial SSR amplitudes for a full day (>5,000 trials). Background EMG was identical for the 2 days. In contrast, the distribution for the SSR↑ day is far to the right of that for the control day. Figure 4*C* shows single-trial raw data from a monkey under the control mode (top) and after prolonged exposure to the SSR↑ mode (bottom). Each trace shows the 40 ms preceding and following pulse onset. Background EMG and the course of elbow extension (lowest trace in each group) are the same under the two modes. Only SSR amplitude is different. It is much greater under the SSR↑ mode.

SSR↓ mode

After collection of control data, six monkeys were switched to the SSR↓ mode for periods of 10–100 days. Background EMG amplitude and the initial 30 ms of pulse-induced extension remained stable. SSR amplitude decreased significantly in five of the six monkeys, while in one no change was noted.[2] As with SSR increase, decrease be-

[2] Subsequent to this single negative result, experience with another animal has indicated that it is occasionally necessary to begin SSR↓ training by rewarding the animal on the basis of a more extensive portion of the

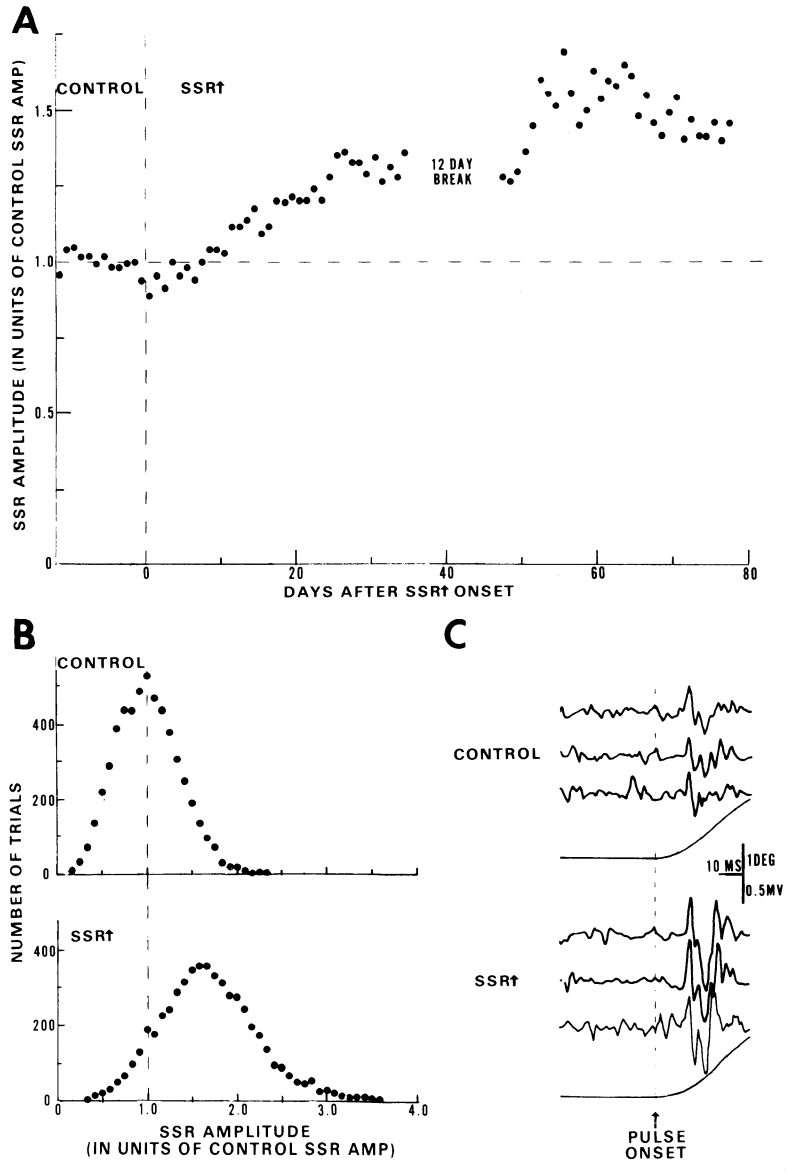

FIG. 4. Effect of prolonged SSR↑ training. *A*: daily SSR amplitudes for the control and SSR↑ periods in one monkey. SSR amplitude is in terms of average SSR amplitude for the control period. SSR increase occurs gradually and does not appear to regress over a 12-day break. *B*: each histogram shows SSR amplitudes for all a monkey's individual trials in a single day (>5,000) in units of average background EMG amplitude, which is the same for both histograms. The top histogram shows a control-period day, the bottom histogram a day after prolonged SSR↑ training. Note marked shift in distribution of SSR amplitudes. *C*: series of individual trials of raw EMG from one monkey under the control mode and after prolonged SSR↑ training. Each series is made up of consecutive trials. Pulse onset is indicated by the vertical dashed line and the average course of pulse-induced extension is shown by the bottom trace. Note that background EMG and pulse-induced extension are the same under both modes. In contrast, SSR amplitude is much greater after prolonged SSR↑ training.

response. As the entire EMG response, including the SSR, begins to decrease, the criterion interval may be rapidly reduced in length until it covers only the SSR. We surmise that had this method been used with the animal under discussion, SSR↓ training would have been successful.

came evident in 5–10 days and progressed over weeks. For the five successful monkeys, final values were 22, 27, 56, 57, and 79% of control. Figure 5*A* shows a monkey's daily SSR amplitude during the control period and

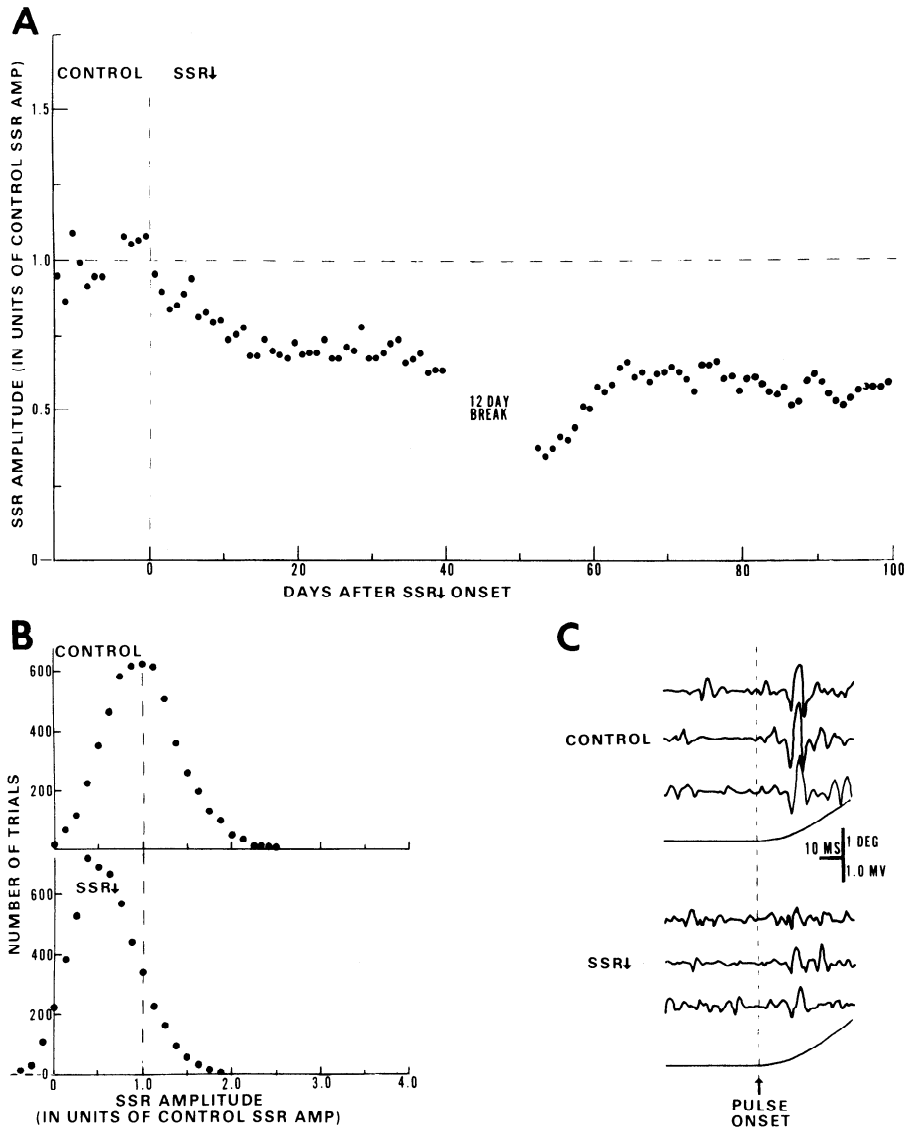

FIG. 5. Effect of prolonged SSR↓ training. *A*: daily SSR amplitudes for the control and SSR↓ periods in one monkey. SSR decrease occurs gradually and does not regress over a 12-day break (see text). *B*: each histogram shows SSR amplitudes for all a monkey's individual trials in a single day (>5,000) in units of average background EMG amplitude, which is the same for both histograms. The top histogram shows a control-period day, the bottom histogram a day after prolonged SSR↓ training. Note marked shift in distribution of SSR amplitudes. (Because SSR amplitude is amplitude in the SSR interval minus average background EMG amplitude, a single trial's SSR amplitude can be less than zero.) *C*: series of individual trials of raw EMG from one monkey under the control mode and after prolonged SSR↓ training. Each series is made up of consecutive trials. Pulse onset is indicated by the vertical dashed line and the average course of pulse-induced extension is shown by the bottom trace. Note that background EMG and pulse-induced extension are the same under both modes. In contrast, SSR amplitude is much smaller after prolonged SSR↓ training.

the SSR↓ period. The SSR decreases markedly over 3–4 wk and appears to continue to decrease slowly over the next several months. The decrease certainly does not regress over a 12-day home-cage break.[3] Figure 5*B* shows

[3] The immediate postbreak SSR decrease and rapid increase are probably an effect of the break itself and not related to mode. A similar postbreak decrease and rapid increase is probably apparent in Fig. 4. This phenomenon will be presented in detail in a subsequent report.

histograms of representative days (>5,000 trials each) before and after SSR↓ onset. After prolonged SSR↓ exposure, the distribution is shifted markedly to the left. Figure 5*C* shows single trials from a monkey before and after SSR↓ onset. Background EMG and the initial 40 ms of extension do not change, while SSR amplitude decreases markedly under the SSR↓ mode.

Biceps II data

We gathered biceps II data primarily to determine whether biceps I behavior was representative of the entire biceps muscle. In the five monkeys for which biceps II data were available, there was a close correlation between biceps I and biceps II behavior throughout data collection. Presumably because its electrodes were closer together (see METHODS), biceps II background EMG averaged 70–80% of biceps I background EMG. For each monkey, this relationship remained constant throughout data collection. The standard deviation of the difference between biceps I and II background EMG was <7% of the biceps I amplitude. Thus, for example, if biceps I background EMG amplitude was 200 μV on a given day, biceps II amplitude was 150 ± 14 μV.

The correlation between biceps I and II SSR amplitudes was also close, as shown in Fig. 6*A*, which summarizes all the data. The slope is +0.92 and r is +0.89 ($P \ll 0.001$). As an example of this correlation, Fig. 6*B* shows biceps I and II raw EMG traces from one monkey under the control and SSR↑ modes.

These close correlations between biceps I and II behavior indicate that background EMG remained stable throughout the biceps muscle and that SSR amplitude change noted in the biceps I EMG occurred throughout the muscle.

Effects on movement

As already noted, for each monkey the course and amplitude of the initial 30 ms of pulse-induced extension remained extremely stable throughout data collection. No change occurred under the SSR↑ or SSR↓ mode. Thus, the stretch eliciting the SSR remained constant. Beyond 40 ms, extension amplitude displayed somewhat more day-to-day variation. A significant part of this variation was attributable to change in SSR amplitude

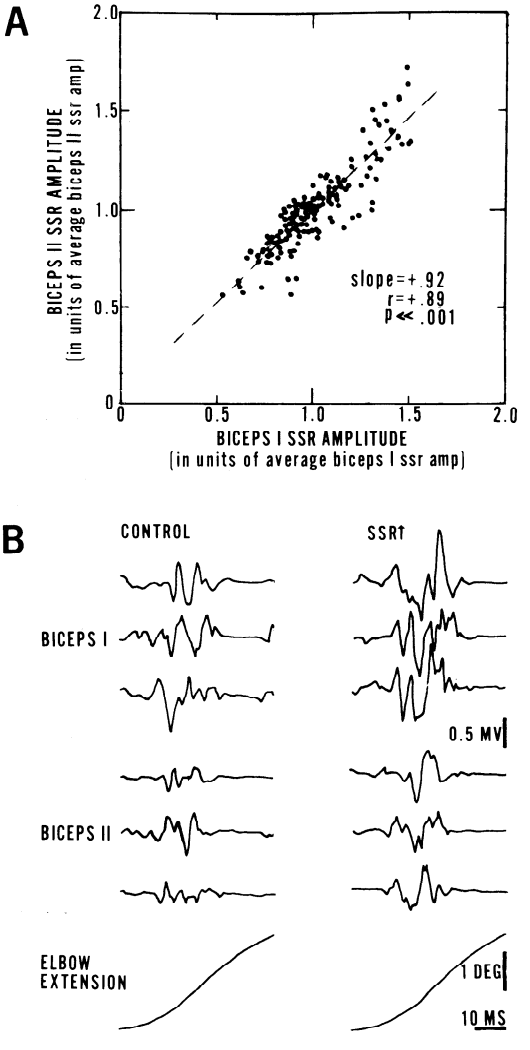

FIG. 6. Comparison of data from biceps I and biceps II electrode pairs. *A*: biceps I SSR amplitude versus biceps II SSR amplitude in all five monkeys for which biceps II data were collected. Each point is a day's data. Biceps I and biceps II SSR amplitudes for a given electrode pair on a given day are expressed in terms of that pair's overall average biceps I and biceps II SSR amplitudes. Biceps I and biceps II are closely correlated (slope = +0.92, r = +0.89, $P \ll 0.001$). *B*: series of individual trials of biceps I and biceps II raw EMG following pulse onset from one monkey under the control mode and after prolonged SSR↑ training. Each series is made up of three consecutive trials during which biceps I and biceps II were both recorded. Bottom traces show average course of pulse-induced extension. Note close correspondence between biceps I and biceps II behavior under the two modes.

induced by the SSR↑ and SSR↓ modes. Figure 7*A* shows linear regression lines of extension at 100 ms versus SSR amplitude for five

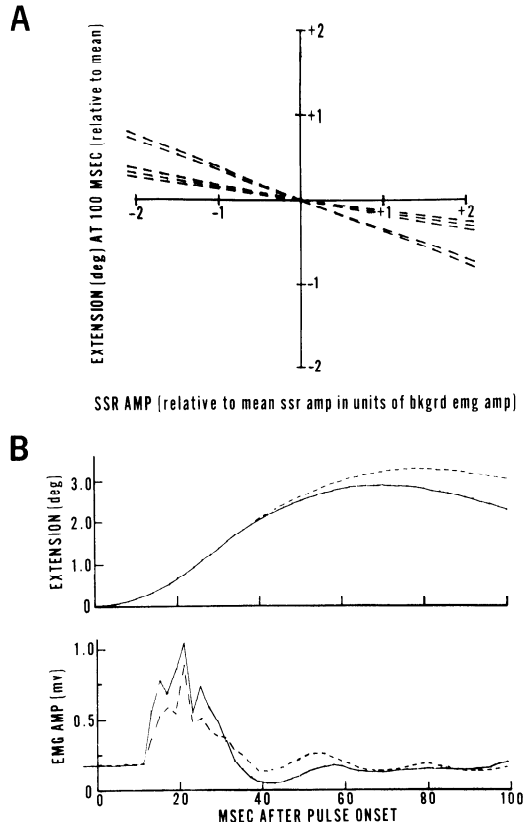

FIG. 7. *A*: elbow extension 100 ms after pulse onset versus SSR amplitude. Linear regression lines are for five monkeys exposed to both SSR↑ and SSR↓ modes so that SSR amplitude varied over a considerable range (2–2.8×). Each line is derived from 64 to 100 days of data from one monkey. All five monkeys show a negative correlation ($r = -0.56$ to -0.88, $P \ll 0.001$) between extension at 100 ms and SSR amplitude. *B*: average elbow extension and absolute value of biceps EMG, beginning with pulse onset, from two representative days in one monkey. Dashed traces are from a day when SSR amplitude was in the control range; solid traces from a day following prolonged SSR↑ training. As in all animals, the initial 30–40 ms of pulse-induced extension is not affected by SSR change, while beyond 40 ms, extension is inversely related to SSR amplitude.

monkeys for which SSR amplitude varied over a considerable range (2–2.8×) due to exposure to both the SSR↑ and SSR↓ modes. For each, extension is negatively correlated with SSR amplitude ($P \ll 0.001$). Figure 7*B* illustrates this correlation with 2 days of data from one monkey. As indicated in this example, SSR amplitude typically begins to affect movement at about 40 ms, 20 ms after the peak of the SSR. This delay is presumably due to the time required for motor-unit contraction following excitation.

DISCUSSION

The data demonstrate that monkeys can markedly change SSR amplitude without change in background α-motoneuron tone, as measured by EMG, and without change in initial muscle length. SSR change occurred without change in the initial pulse-induced stretch, without visible change in arm posture, daily performance schedule, or overt animal behavior, and without change in SSR latency. The SSR could be nearly doubled by the SSR↑ mode or quartered by the SSR↓ mode. Because SSR amplitude was stable during prolonged control recordings and because change under the SSR↑ or SSR↓ mode was appropriate to the mode, we conclude that amplitude change was a specific adaptive response to a specific external condition. It seems reasonable to assume that SSR plasticity is not unique to experiments like the present one. While SSR amplitude may not always have as significant an effort on limb position as in Fig. 7 and while the primary function(s) of the SSR remain uncertain (27), the SSR is clearly a significant factor in motor performance. Adaptive SSR amplitude changes, helping to optimize performance, probably occur in the normal course of growth and aging and in response to demands of many sorts. Evidence (56) that SSR change is relatively specific to the agonist muscle supports this assumption.

The first issue raised by our results is the mechanism of the observed change. While the mechanism is certainly activated by descending input caused by the monkey's desire for a greater number of rewards, it must reside somewhere in the segmental arc of the SSR. A second issue concerns the purpose of the study, development of a system allowing investigation of the neuronal and synaptic bases of memory. The time course of SSR change, the long-term continual performance necessitated by the task eliciting it, and a considerable body of related clinical and experimental evidence strongly suggest that the experiment may in fact have produced an enduring segmental change.

Possible mechanisms of SSR amplitude change

The response to sudden muscle stretch begins with excitation of peripheral receptors. While a sudden extension, such as that of the present study, excites a variety of deep and superficial limb receptors, muscle spindle excitation is largely responsible for the initial, purely segmental, response of the stretched muscle (36). Furthermore, in the absence of change in limb posture or in initial muscle length or tone, only the muscle spindle among sensory receptors can undergo significant change in sensitivity to stretch and thus alter SSR amplitude. Spindle sensitivity is affected by γ-motoneuron tone. In isometric situations similar to the present experiment, γ-motoneuron tone has been found to be closely linked to α-motoneuron tone (37, 50), which was here kept constant as measured by EMG. However, the possible effects of long-term, continual task exposure, such as that required by the present experiment, have not previously been explored. Pathways capable of independently altering α- and γ-motoneuron tone certainly exist, and dissociation has been described in other circumstances (27, 49). Even if α-γ linkage was preserved in our study, it is conceivable on the basis of the data presented here that change in the activity of synergist motoneurons could have altered the sensitivity of their spindles. (However, further data, presented in the following paper (56), indicate that background EMG in synergist and antagonist muscles did not change with biceps SSR amplitude.) The work of Eldred et al. (16), and Hunt (28) and more recent work (4, 44) indicate that spindle sensitivity might also be changed by sympathetic influences. Thus, at present, change in muscle spindle sensitivity must be considered a reasonable possibility for the mechanism of the observed change in SSR amplitude. However, it is relevant to note that change in muscle spindle sensitivity does not seem to be the mechanism underlying either the Jendrassik maneuver (7, 10) or vibratory inhibition of the SSR (2).

The initial agonist α-motoneuron response to muscle stretch is produced largely, though probably not exclusively, by monosynaptic input (3, 24, 36). The major portion of this input is via Ia-fibers. While monosynaptic connections from group II spindle fibers have been described, these fibers are less sensitive to sudden stretch than Ia-fibers, and their monosynaptic input is considerably weaker and less widely distributed (48). In the present study, the SSR interval, as defined by the computer, ended at 24 ms, thus excluding the last portion of the SSR, which is somewhat more likely to be affected by polysynaptic segmental pathways. The Ia synapse on the α-motoneuron is subject to presynaptic inhibition via pathways originating both peripherally and centrally (3, 9, 34). Vibratory inhibition of the SSR is almost certainly mediated by this means (2). Increased presynaptic inhibition could be responsible for the effect of the SSR↓ mode, while reduction could be responsible for the effect of the SSR↑ mode. Activity in group II and III afferents, for example, may alter Ia synaptic transmission (3, 33). However, in the absence of any visible change in arm position or posture, it is unlikely that change in such input would occur and affect Ia synaptic function so as to significantly change SSR amplitude. The absence of change in synergist and antagonist muscle activity, documented in the following report (56), makes it still less likely. If change in presynaptic inhibition is in fact the mechanism of SSR amplitude change, it seems most probable that descending pathways are responsible.

The α-motoneuron itself is the next station in the reflex arc and the next possible site of change. During the prepulse, isometric portion of the task, biceps α-motoneurons presumably comprise a continuum ranging from rapidly firing cells, through more slowly firing cells, through cells just below firing threshold, to cells well below firing threshold (24). Any substantial change in central or peripheral input to this population would change the proportion of cells that are firing and affect firing rates, and thereby affect the level of background EMG. But the task required that background EMG remain at a given level; thus it prevented any such overall change in biceps α-motoneuron excitability. However, several more specific and subtle α-motoneuron mechanisms must still be considered as possible contributors to SSR amplitude change. First, localized postsynaptic changes in dendritic geometry or membrane properties might alter the neuron's responsiveness to Ia input

(26, 46). Second, α-motoneuron recruitment order might change, perhaps by change in input resistance (57), so that a different segment of the population is near firing threshold at the time of muscle stretch. Because sensitivity to Ia input is different for different α-motoneurons (9), such a change could alter SSR amplitude. While recruitment order appears to be relatively fixed in most situations (24), significant changes can occur (8). However, smaller motoneurons, which are normally recruited first and should thus be active under the control mode, are most sensitive to Ia input (24). Thus, while change in recruitment order might account for SSR decrease under the SSR↓ mode, it is difficult to conceive how it could account for SSR increase under the SSR↑ mode. Third, since EMG as recorded here is a product of the overlap of many bipolar motor-unit potentials, its amplitude is determined not only by the number and size of the individual motor-unit potentials but by their relative timing. Thus, slight changes in relative α-motoneuron firing latencies might conceivably change SSR amplitude as measured by the EMG electrodes without change in actual motoneuron firing.

The design of the experiment makes it very unlikely that change in neuromuscular coupling or in the relationship between motor-unit excitation and the resulting EMG potential could have been responsible for the SSR amplitude changes observed. Changes in either of these factors would have similarly affected background EMG amplitude and SSR amplitude. However, the task prevented change in background EMG amplitude. Thus, the only way in which neuromuscular or muscular change could have altered SSR amplitude would be if it had been accompanied by a marked alteration in the population of motoneurons responsible for the unchanged background EMG amplitude and if this new motoneuron population responded differently to muscle stretch than the original population. As noted above, substantial change in the composition of the active motoneuron population via change in recruitment order probably could not account for all the data. Substantial change in the total number of motoneurons responsible for the background EMG was probably unlikely. Furthermore, our observations and those of others (12) indicate that the measure of SSR amplitude used here, the ratio of SSR amplitude to background EMG amplitude, is relatively stable over a considerable range of background EMG amplitude, that is, in spite of considerable change in the number of active motoneurons. Finally, muscle stiffness, as monitored by the initial 30 ms of pulse-induced stretch, did not change. Therefore, substantial change in motor-unit properties or in the active motoneuron population probably did not occur.

Thus, at present, the most likely site(s) of the mechanism(s) responsible for SSR amplitude change seem to be the Ia synapse and/or the muscle spindle. Our current studies investigating H-reflex amplitude during SSR amplitude change and the slope of SSR amplitude versus stretch amplitude before and after training should contribute to the resolution of this issue. Whether the mechanism of adaptive SSR amplitude change is related to that of the much more modest diurnal variation in SSR amplitude (32, 55) remains unclear. Current evidence indicates that the diurnal variation changes little in phase or magnitude with imposition of the SSR↑ or SSR↓ mode (unpublished data). The relative specificity of SSR amplitude change to the agonist muscle, documented in the following paper (56), also suggests that the mechanisms are different.

Long-term segmental change

The impetus for adaptive SSR amplitude change certainly originated suprasegmentally. In the initial stages of SSR↑ or SSR↓ training, this impetus was doubtless simply imposed on an otherwise naive segmental apparatus. However, the task design ensured that the monkey did not know exactly when the sudden muscle stretch would occur, and once it did occur the SSR was over well before any other possible reaction to it. Thus, if the descending influence changing SSR amplitude was to be effective, it had to be present for a considerable proportion of the 5–7 h/day each animal spent working. This situation persisted throughout the many days of SSR↑ or SSR↓ training. A considerable body of clinical and experimental evidence strongly suggests that such long-term descending influence will eventually produce persistent segmental

changes no longer dependent on that descending influence.

DiGiorgio (14) demonstrated that the asymmetric hindlimb posture produced by a hemicerebellar lesion persisted after thoracic cord section if at least 45 min passed between the cerebellar lesion and cord section. Subsequent studies (1, 11, 35) confirmed this result, showed that it occurred with a variety of suprasegmental lesions, and indicated that the responsible changes resided in the cord itself rather than in the peripheral apparatus. Cord transection initiates an as yet incompletely described sequence of changes in segmental function that continue to develop over many months (6, 38, 40, 41). Changes in the cord as well as in the periphery contribute to this progression. For example, recent studies (42) of Ia EPSPs in α-motoneurons below cord transection show that substantial changes in EPSP size evolve over months. Finally, studies over the last several decades indicate that the isolated spinal cord has the capacity for classical conditioning (45).

These data indicate that, given the correct impetus for sufficient time, the cord has the capacity for persistent change. They suggest that such a change may have occurred in the present study due to chronic exposure to the SSR↑ or SSR↓ mode. The salient features of SSR amplitude change, that it takes days to become apparent, continues to progress over weeks, and persists over 12-day gaps in training, appear to support this possibility. The time course of development (and of reversal and redevelopment (53)) is even slower than that of adaptive plasticity in the vestibulooccular reflex (39), which is also thought to involve persistent change at or near the level of a reflex arc. Thus, our current work is directed not only at further defining SSR amplitude change and its mechanism but at determining whether it does involve persistent segmental change, change that can endure without continuing descending influence. If such intrinsic segmental change occurs, it constitutes a fragment of a memory accessible to physiologic and anatomic investigation on the neuronal and synaptic levels with presently available techniques.

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