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Adaptive Plasticity in Primate Spinal Stretch Reflex: Behavior of Synergist and Antagonist Muscles

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SUMMARY AND CONCLUSIONS

1. Monkeys can gradually change the amplitude of the biceps spinal stretch reflex (SSR) without change in initial muscle length or biceps background electromyographic activity (EMG) (17). We investigated the concurrent behavior of synergist (brachialis and brachioradialis) and antagonist (triceps) muscles.

2. Synergist background EMG remained stable while marked change occurred in biceps SSR amplitude. Triceps background EMG was minimal under all conditions. Thus biceps SSR amplitude change was not due to change in the background activity of closely related muscles.

3. When biceps SSR amplitude changed, synergist SSR amplitude changed similarly but to a lesser extent. Brachialis change averaged 72% of biceps change, while brachioradialis change averaged 33%. By indicating that SSR amplitude change is relatively specific to the agonist muscle, this finding eliminates a number of nonspecific mechanisms as possible origins of SSR amplitude change. Thus it supports the potential value of the SSR as a system for studying the neuronal and synaptic bases of memory in the primate central nervous system (CNS).

INTRODUCTION

The preceding paper (17) demonstrated that monkeys can gradually change the amplitude of the earliest, purely segmental, response to sudden biceps stretch, the spinal stretch reflex (SSR), without change in initial muscle length or in background EMG activity. These results suggest that the SSR may furnish a system for studying the neuronal and synaptic bases of memory in the primate CNS. The present study investigated the concurrent behavior of synergist and antagonist muscles in order to answer two important questions.

The first question was whether biceps SSR change is accompanied by change in synergist and/or antagonist background activity. The initial study (17) demonstrated biceps SSR change without change in biceps background EMG. However, change in synergist background activity might have occurred, perhaps accompanied by compensatory change in antagonist activity. Such synergist change would presumably be accompanied by change in sensitivity of synergist muscle spindles, which may have substantial monosynaptic access to biceps motorneurons (1, 4, 7, 11, 15). Thus, change in synergist muscle background activity might account for the observed change in biceps SSR amplitude, thereby rendering biceps SSR change a less interesting phenomenon.

The second question was whether comparable SSR amplitude change occurred in synergist muscles. The initial study (17) indicated that SSR amplitude change occurred throughout the biceps muscle. Simultaneous, comparable SSR change in other muscles would compel consideration of a number of nonspecific mechanisms (see DISCUSSION) and could reduce the value of SSR change as a system for studying CNS adaptive mechanisms.

METHODS

Our standard animal preparation and training methods have been fully described (16, 17) and are simply summarized here, with emphasis on procedures relating to synergist and antagonist muscles.

Animal preparation and training

Subjects were six monkeys (*Macaca nemestrina,* male, 5–7 kg). Ten stainless steel fine-wire EMG electrodes (3) were chronically implanted in several arm muscles. Two pairs of electrodes, a primary pair (biceps I) and a secondary pair (biceps II), were implanted in biceps, and single pairs were implanted in the synergists, brachialis and brachioradialis, and the antagonist, triceps. (Biceps data reported here are from biceps I, except as noted. For detailed biceps II data, see Ref. 17.) One of the six monkeys had only biceps and triceps pairs.

After surgery, each monkey was seated with the forearm resting in a cast attached at the elbow to a torque motor shaft. The motor applied constant modest (ca. 0.5 nm) elbow extension torque. Working for a liquid reward, the animal learned a two-part computer-controlled task. It learned to keep elbow angle at 90° (±1.5°) against the steady extension torque for a period varying randomly from 1.2 to 1.8 s, and to keep the average absolute value of biceps EMG (equivalent to full-wave rectified EMG) for the final 200 ms within a specific range. If it accomplished this two-part task, a brief pulse of additional extension torque transiently extended the elbow 2–3° and elicited the biceps SSR. The computer calculated the average absolute value of the biceps EMG in the SSR interval, normally defined as 14–24 ms after stretch onset. Under the control mode, reward occurred 200 ms after pulse onset. Under the SSR↑ or SSR↓ mode, reward occurred only if EMG amplitude in the SSR interval was greater than (SSR↑) or less than (SSR↓), a criterion value. Monkeys usually completed 3,000–6,000 trials daily. The computer gave a daily summary, including average background (i.e., prepulse) biceps EMG amplitude, average initial elbow angle, and average course of biceps EMG amplitude and elbow angle following pulse onset. Daily biceps SSR amplitude was defined as the average EMG amplitude in the SSR interval minus average background EMG amplitude. In addition, raw EMG and elbow angle data were recorded on analog tape periodically.

Synergist and antagonist muscle data

As noted above, the computer always monitored elbow angle and the biceps I electrode pair. In addition, it simultaneously monitored at least one additional pair of electrodes (biceps II, brachialis, brachioradialis, and/or triceps). On each occasion, a full day's data were obtained from the additional pair(s). Thus, each of these four pairs was monitored at least every 4th day.

In four monkeys these additional pairs were simply monitored, no attempt was made to control background EMG in the synergist and/or antagonist muscles. In the other two monkeys, following software and hardware expansion, three to five EMG channels were monitored simultaneously, and background EMG limits were placed on the brachialis muscle as well as on the biceps. In all monkeys in the SSR↑ and SSR↓ modes, reward was contingent only on biceps I SSR amplitude, never on synergist SSR amplitude.

RESULTS

Data were collected from each animal over 3–15 mo. Animals were closely monitored throughout (16, 17). They remained healthy and active. First, each monkey worked under the control mode for 10–30 days. Then, either the SSR↑ or SSR↓ mode was imposed for at least 35 days. For the remainder of data collection, animals underwent sequences involving mode reversal (SSR↑ to SSR↓ or visa versa), return to the control mode, and/or removal from the task for periods of up to 31 days.

Throughout data collection, biceps background EMG and the initial 30 ms of pulse-induced extension remained stable (17). The behavior of the biceps II electrode pair closely paralleled that of the biceps I pair (17). In all animals, biceps SSR amplitude changed appropriately with imposition of the SSR↑ or SSR↓ mode. Change became apparent over 5–10 days and progressed over weeks.

Synergist and antagonist background EMG under control mode

Brachialis background EMG amplitude was comparable to that of biceps. Brachioradialis background was somewhat less, averaging 50–75% of biceps I. In the two monkeys in which brachialis (as well as biceps) background EMG criteria were imposed, brachialis background EMG remained stable throughout the control period. Brachioradialis background EMG in these monkeys and brachialis and brachioradials background EMG in the other monkeys showed somewhat more variation. The standard deviation of daily values averaged 10–15% of the average value for the entire control period.

Antagonist (triceps) background EMG was

very low in all animals, averaging about 10% of biceps or brachialis background EMG (Fig. 1). It was so low that a substantial portion of it may in fact have been activity from biceps or other flexors picked up at a distance by the triceps electrodes. This minimal activity remained stable throughout the control periods.

Synergist and antagonist background EMG under SSR↑ and SSR↓ modes

Synergist background EMG did not change following imposition of the SSR↑ or SSR↓ mode. Control period values persisted throughout the course of change in biceps SSR amplitude. Figure 1, which displays one monkey's data, shows progressive SSR increase under the SSR↑ mode without significant change in agonist, synergist, or antagonist background EMG. Figure 2 presents all the data from the five monkeys with synergist EMG electrodes. It is clear that synergist background EMG in these monkeys did not display the significant positive correlation with biceps SSR amplitude that might be expected if synergist muscle spindle sensitivity change was the origin of biceps SSR change. A slight, insignificant negative correlation is present (slope = −0.26, $r = -0.16$, $P = 0.10$).

As illustrated in Fig. 1, triceps background EMG remained very low under the SSR↑ and SSR↓ modes, displaying no change from its control period behavior.

These data prompt two conclusions. First, SSR amplitude changes produced by the SSR↑ and SSR↓ modes cannot be attributed to changes in synergist or antagonist background activity. Second, moderate variation in synergist background activity, occurring mainly in those animals without brachialis background EMG criteria, had no significant effect on biceps SSR amplitude (Fig. 2).

Synergist SSRs under control mode

Under the control mode, brachialis and brachioradialis SSRs were comparable in latency and amplitude to that of biceps and remained similarly stable. Very small responses of SSR latency were usually evident in the triceps (Fig. 4). However, as noted above, it was not clear whether this was in fact purely triceps activity or was due to contamination by the much higher amplitude flexor muscle responses.

Synergist SSRs under SSR↑ and SSR↓ modes

When biceps SSR amplitude changed under the impetus of the SSR↑ or SSR↓ mode,

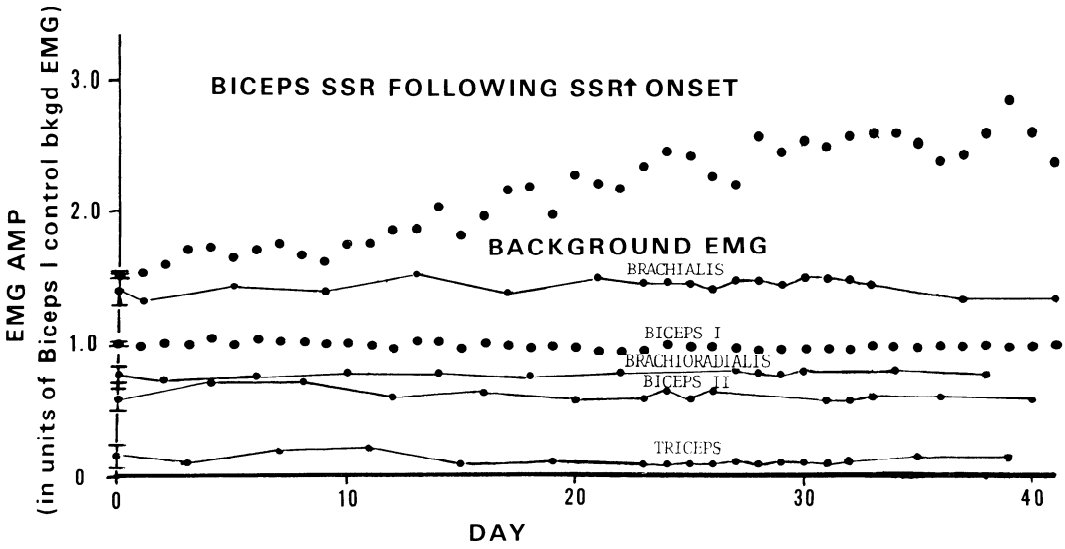

FIG. 1. Daily agonist (biceps I and biceps II), synergist, and antagonist background EMG following imposition of the SSR↑ mode in one monkey. All values are in terms of average biceps I background EMG. SSR amplitude rises steadily. In contrast, background EMG in biceps, in its synergists, brachialis and brachioradialis, and in its antagonist, triceps, does not change. Note that electrode pairs other than biceps I were monitored 1 of 4 days or more often.

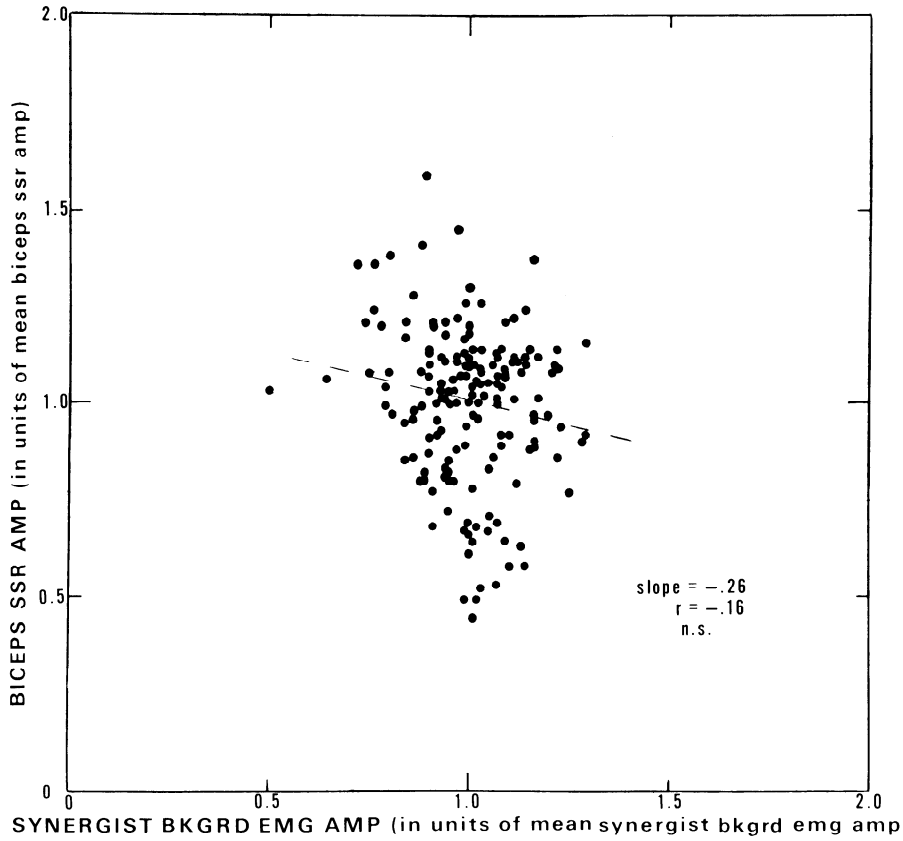

FIG. 2. Biceps SSR amplitude versus synergist background EMG amplitude for the five monkeys in which brachialis and brachioradialis background EMG was monitored. Each point represents average biceps SSR amplitude and average sum of brachialis and brachioradialis background amplitudes for a 2-day period. Since brachialis background was normally greater than brachioradialis background, brachialis has a greater effect on the plotted value. Linear regression line shows a slight nonsignificant negative correlation (slope = −0.26, r = −0.16, P = 0.10).

synergist SSRs underwent similar but lesser change. Figure 3 shows the progressive changes in SSR amplitudes occurring in the Fig. 1 monkey following onset of the SSR↑ mode. SSR increase is most marked for the biceps, less marked for the brachialis, and still less marked for the brachioradialis. Figure 4 illustrates this relationship with individual trials of raw EMG. The biceps SSR increases markedly. The brachialis SSR increases slightly. The brachioradialis SSR shows no apparent change. Figure 5 summarizes the data from all five monkeys with synergist EMG electrodes. Brachialis SSR change averages 72% of biceps change, brachioradialis averages 33%. In sum, SSR amplitude change was greatest in the biceps muscle. It also occurred to a considerable but lesser extent in the brachialis, and to a modest degree in the brachioradialis. Thus it was relatively, though not completely, specific to the agonist muscle.

As noted above, triceps responses in the SSR latency range were very small (Fig. 4) and of uncertain origin. They displayed no significant or consistent change when biceps SSR amplitude changed.

DISCUSSION

The mechanism of adaptive change in SSR amplitude must reside somewhere in the segmental arc of the SSR. It could be either a result of continuing descending influence or a result of persistent segmental change (17). At present, the two most likely sites appear

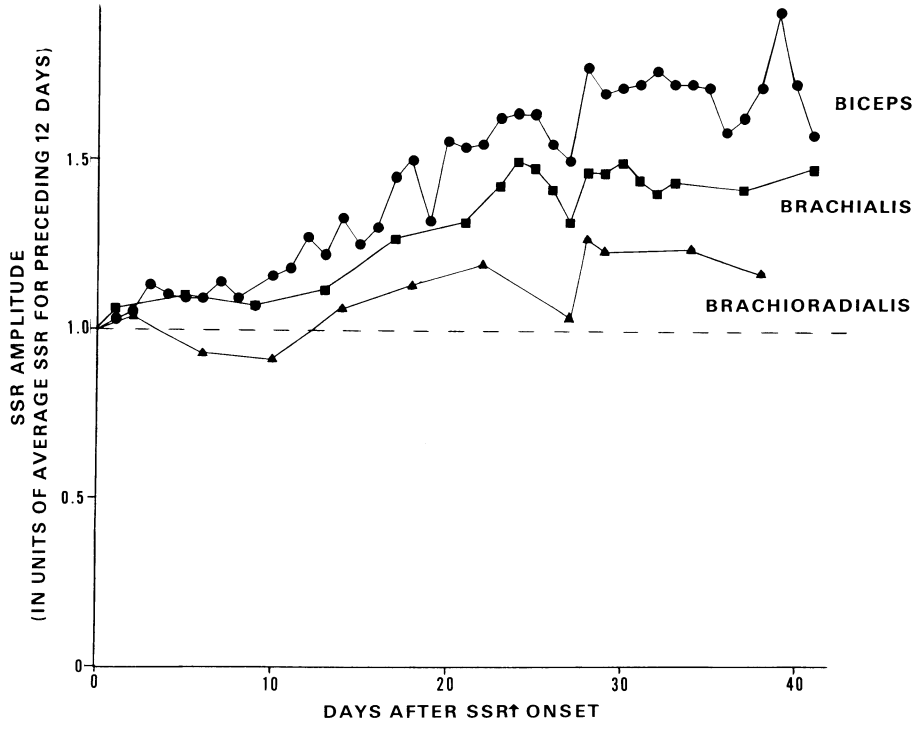

FIG. 3. Biceps, brachialis, and brachioradialis daily SSR amplitudes in the Fig. 1 monkey following onset of SSR↑ mode. Each muscle's SSR amplitude is in terms of its average SSR amplitude for the previous 12 days. Note marked increase in the biceps SSR, smaller increase in the brachialis SSR, and still smaller increase in the brachioradialis SSR.

to be the muscle spindle and the Ia-afferent fiber synapse on the α-motoneuron (17). Change in muscle spindle sensitivity would change the amplitude of the Ia-afferent volley produced by sudden extension, while change in Ia synaptic function would alter the volley's effect on the α-motoneuron.

Muscle spindle sensitivity is significantly affected by γ-motoneurons (12) and probably by other factors, including sympathetic fibers (2, 6, 9, 13). In situations similar to the present experiment, γ-motoneuron tone has been found to be closely linked to α-motoneuron tone, while in other situations dissociation has been observed (14). Because biceps α-motoneuron tone remained stable in the present experiment, while SSR amplitude changed, γ-motoneurons controlling biceps spindles could have been responsible for SSR change only if α-γ dissociation occurred. Though data from the monkey upper arm are not available, related data (1, 4, 7, 11, 15) suggest that spindles in synergist muscles may be expected to have significant Ia monosynaptic effects on biceps α-motoneurons. Thus, monkeys might change biceps SSR, without α-γ dissociation, by simply changing synergist muscle tone. If this occurred, it would constitute an essentially trivial explanation for the SSR amplitude changes we have observed (17). But the present study demonstrates that significant and/or appropriate change in synergist background EMG did not accompany SSR amplitude change. Thus, the biceps SSR did not increase simply because synergist α-γ tone increased or decrease because this tone decreased. If synergist (or agonist (17)) γ-motoneuron tone was in fact responsible, then it must have involved α-γ dissociation.

It is particularly striking that synergist background EMG showed no consistent or marked changes on imposition of the SSR↑ or SSR↓ mode even in those monkeys in

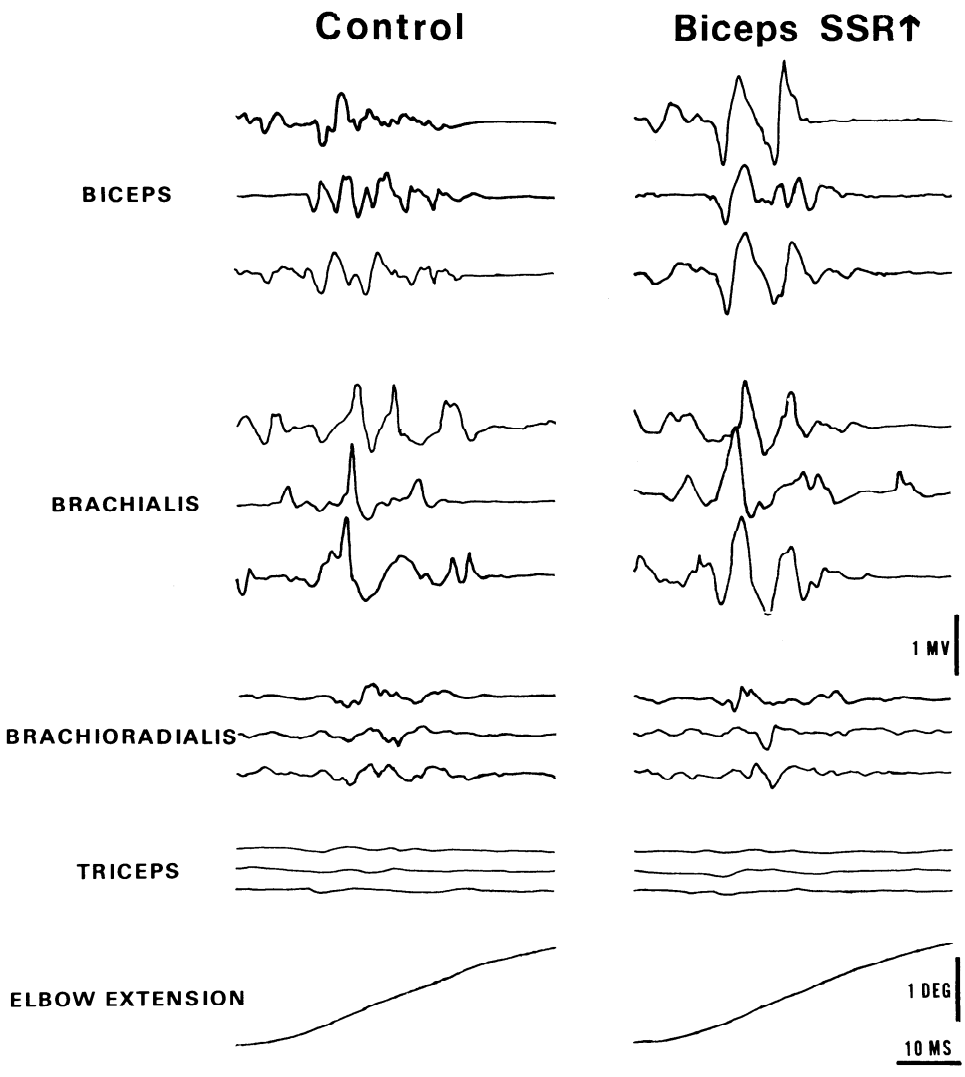

FIG. 4. Biceps, brachialis, brachioradialis, and triceps raw EMG from individual trials under the control mode (left) and after prolonged SSR↑ training (right) in one monkey. Bottom traces show average course of pulse-induced extension. It is the same under both modes. Note marked biceps SSR increase after SSR↑ exposure, modest brachialis SSR increase, and apparent absence of brachioradialis SSR increase. Background EMG, represented here by the first 10 ms following pulse onset, is the same under both modes. Triceps activity is minimal throughout.

which only biceps background EMG was controlled. This finding, combined with the fact that the changes in synergist background EMG that did occur had little or no apparent effect on biceps SSR amplitude, suggests that heteronymous effects are relatively weak. Thus it is less likely that alterations in the behavior of more remote muscles, such as others in the forearm, played a role in SSR amplitude change. Furthermore, the fact that forearm, hand and shoulder postures were stable throughout data collection (17) made such alterations less likely.

The data indicate that the biceps SSR amplitude changes induced were relatively specific. They were most marked in the biceps. Less change occurred in the SSR of the close synergist, brachialis, and still less in that of the somewhat more remote synergist, brachioradialis. It is important to note that this

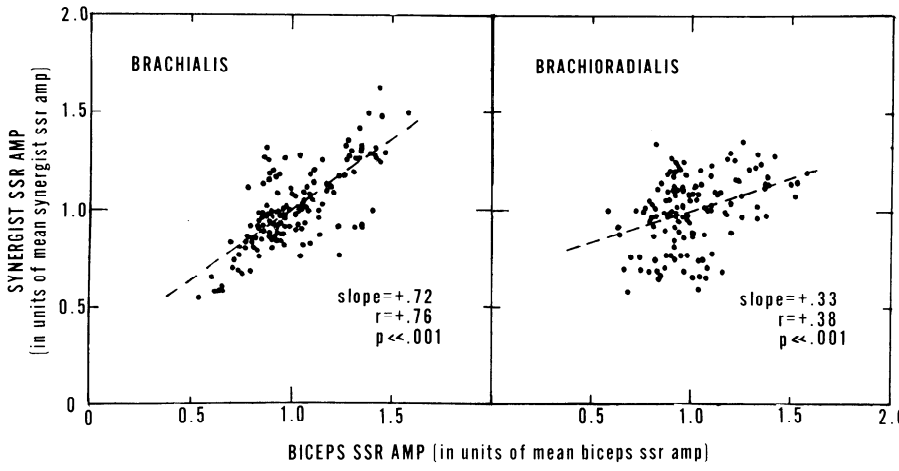

FIG. 5. Biceps SSR amplitude versus brachialis SSR amplitude (left) and versus brachioradialis SSR amplitude (right) in the five monkeys in which synergist muscles were monitored. Each point is a day's data from one monkey. Each day's biceps and synergist SSR amplitudes are in terms of the monkey's average biceps and synergist SSR amplitudes over the entire data-collection period. Biceps and brachialis are closely correlated ($r = +0.76$, $P \ll 0.001$). Brachialis SSR change averages 72% of biceps SSR change. Biceps and brachioradialis are somewhat less closely correlated ($r = +0.38$, $P \ll 0.001$). Brachioradialis change averages only 33% of biceps change. Thus effects of SSR↑ or SSR↓ training are most marked in the agonist muscle, weaker in a close synergist, and still weaker in a more distant synergist.

relative specificity was not required by the task, reward was in no way contingent on synergist SSR amplitude. Thus its occurrence is more noteworthy. We might well have obtained greater specificity had the task required it (for example, reward only if biceps SSR > a criterion value and brachialis SSR < a criterion value). The observed specificity provides little clue as to the location in the reflex arc of the mechanism responsible for SSR change. It does, however, allow us to rule out with some confidence a variety of nonspecific mechanisms that would be expected to affect many muscles or the entire body. Thus, SSR change was probably not produced by increase in remote muscle tone, that is, by a Jendrassik maneuver (5, 10). Nor was it produced by a diffuse change in sympathetic tone or in concentration of a circulating sympathetic factor, either of which might have been expected to lead to widespread change in muscle spindle sensitivity (2, 6, 9, 13). Similarly, SSR change cannot easily be attributed to any changes in muscle bulk or fiber type occurring as a result of the prolonged task exposure (17). In sum, the data indicate that SSR amplitude change was a relatively focal as well as adaptive event; and thus it is of greater interest as a possible system for studying the neuronal and synaptic bases of memory in a primate (17).

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