JOURNAL OF NEUROPHYSIOLOGY
Vol. 55, No. 2, February 1986. *Printed in U.S.A.*

Adaptive Plasticity in Primate Spinal Stretch Reflex: Persistence

JONATHAN R. WOLPAW, JULIE A. O'KEEFE,
PATRICIA A. NOONAN, AND MICHAEL G. SANDERS

Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, 12201; and Departments of Neurology and Anatomy, Albany Medical College, Albany, New York 12208

SUMMARY AND CONCLUSIONS

1. Monkeys can gradually change the amplitude of the wholly segmental, largely monosynaptic, spinal stretch reflex (SSR) when confronted by a task requiring such change (15–19). Change develops over months and may reverse and redevelop at similarly slow rates. We investigated the persistence of SSR amplitude change over nonperformance periods of up to 38 days.

2. Eight animals with chronic EMG electrodes learned to maintain elbow angle and a given level of biceps background EMG against constant extension torque. At random times, a brief additional extension torque pulse elicited the biceps SSR. In the control mode, reward always followed. Under the SSR↑ or SSR↓ mode, reward occurred only if the absolute value of biceps EMG in the SSR interval was above or below a set value. Animals completed 3,000–6,000 trials/day over data-collection periods of 2–17 mo.

3. Animals worked first under the control mode for up to 60 days and then under the SSR↑ or SSR↓ mode for up to 274 days. Mode was switched once or twice more (SSR↑ to SSR↓ or vice versa) over subsequent months. Animals responded to each SSR↑ or SSR↓ mode exposure with gradual mode-appropriate change in SSR amplitude. Mode exposures were interrupted by gaps in performance of 10–38 days.

4. Gaps produced transient 10- to 15% decreases in SSR amplitude under the control mode. This nonspecific decrease disappeared over the first week of postgap performance. Under the control mode, gaps had no other effects on SSR amplitude.

5. Under the SSR↑ mode, gaps usually produced substantial decline of the adaptive SSR increase from its pregap value. Thus, in the absence of performance, SSR increase appeared to decay with a half-life of ~17 days.

6. Under the SSR↓ mode, gaps had no consistent effect on adaptive SSR decrease. Thus, in the absence of performance, SSR decrease usually persisted without significant change for at least 1 mo.

7. The data showed no evidence of rapid adaptive change in the early postgap period. SSR behavior in the postgap period was a combination of recovery from the modest nonspecific gap-induced decrease and resumption of slow mode-appropriate adaptive change.

8. These results are consistent with the hypothesis that adaptive SSR change involves persistent structural and/or biochemical alterations and thus may provide a technically accessible substrate of memory.

INTRODUCTION

Investigation of the substrates of vertebrate memory requires a stimulus-response pathway that satisfies three crucial requirements: anatomic and physiological definition and accessibility, capacity for displaying adaptive change, and possession of the responsible substrate (14, 16). Most stimulus-response pathways in the vertebrate central nervous system (CNS) are intricate, incompletely delineated, and not easily accessible, rendering it exceed-

ingly difficult to localize and define changes. Furthermore, since neurons in these pathways receive input from other CNS areas, any changes noted might simply be due to tonic inputs originating elsewhere.

Our current studies are directed at developing a suitable model based on the simplest, best defined, most accessible, and scientifically most productive stimulus-response pathway in the intact vertebrate CNS: the two-neuron monosynaptic arc largely, though not exclusively (3), responsible for the initial response to sudden muscle stretch (6, 9), referred to here as the *spinal stretch reflex* (SSR). We selected this pathway because it is the only one in the vertebrate CNS that rivals invertebrate pathways in anatomic and physiological delineation and technical accessibility and because a considerable body of clinical and experimental evidence suggested that it might be capable of long-term adaptive change (16). Initial studies (15–17) demonstrated that monkeys can gradually change SSR amplitude without change in initial muscle length or background α-motoneuron activity as measured by EMG, when reward is contingent on amplitude. Amplitude change occurs over months. Reversal and redevelopment, motivated by alteration in the reward contingency, also occur gradually. SSR amplitude change is relatively specific to the agonist muscle and affects movement. Detailed analysis of the course of SSR amplitude change (18, 19) indicates that it occurs in two phases: an immediate small change (phase I), presumably due to operantly conditioned alteration in tonic descending spinal cord activity, and very slow prolonged change (phase II), suggesting gradual phase I-induced structural or biochemical alterations. The magnitude and other features of SSR change strongly suggest involvement of the monosynaptic arc (16). Thus the SSR pathway, which fulfills the first requirement of definition and accessibility, also fulfills the second, capacity for long-term adaptive plasticity, and may satisfy the third, possession of the responsible substrate. SSR amplitude change appears to persist for considerable periods in the absence of continued task performance. The degree and duration of this persistence are important features of the phenomenon, since they bear on the possibility that SSR change involves persistent alterations. This study evaluates in detail the persistence of SSR amplitude change over nonperformance periods ranging from 10 to 38 days.

METHODS

Experimental techniques, fully described elsewhere (16), are summarized here. Eight monkeys (*Macaca nemestrina*, male, 5–7 kg) were prepared under general anesthesia with chronic intramuscular stainless steel fine-wire EMG electrodes (2) in one biceps muscle and in its antagonist, triceps, and its synergists, brachialis and brachioradialis. Such electrodes remain in place and provide stable data indefinitely. Wires passed subcutaneously through a protected exit in the forearm to a small connector plug. EMG was amplified ($\times$1,000, band pass 10–2,000 Hz) and digitized (2,000 Hz).

Each animal sat with forearm resting in a cast attached at the elbow to a torque motor shaft as shown in Fig. 1*A*. Positions of hand, forearm, arm, and shoulder were controlled. Monitored and rewarded by computer, the animal learned the task outlined in Fig. 1*B*. It learned first to keep elbow angle at 90° ($\pm$1.5°) against a constant moderate extension torque (0.5 N · m) furnished by the motor. If correct angle was held for a randomly selected 1.2- to 1.8-s period *and* if the average absolute value of biceps EMG (equivalent to full-wave rectified EMG) for the final 0.2 s was in a specified range, the motor delivered a short pulse of additional extension torque, which transiently extended the elbow and elicited the biceps SSR. The computer digitized biceps EMG (2,000 Hz) and elbow angle (500 Hz) for 100 ms following pulse onset. It calculated the average of the absolute values of the digitized biceps EMG over the SSR interval (usually 14–24 ms after pulse onset). Liquid reward was given 200 ms after pulse onset. Thus the animal held correct elbow angle and correct biceps EMG for a randomly chosen period, felt the slight extension pulse, opened its mouth, and received the reward squirt.[1] The task functioned under one of three modes. Under the control mode, reward always followed the pulse. Under the SSR↑ or SSR↓ mode, reward occurred only if the average absolute value of biceps EMG during the SSR interval was greater (SSR↑) or less (SSR↓) than a specified value. On initiation of the SSR↑ or SSR↓ mode, the specified value was selected on the basis of control-mode SSR amplitude so as to reward 50–55% of the trials. As SSR amplitude changed and reward percentage consequently moved into the 60- to 70% range, we repeatedly changed the specified value to reduce percentage to 50–55%. Thus the animal never ap-

[1] In 2 of the 8 animals, the primary implanted muscle was the triceps rather than the biceps, so that flexion background torque and a flexion torque pulse were used. Procedures were otherwise identical, and results were comparable.

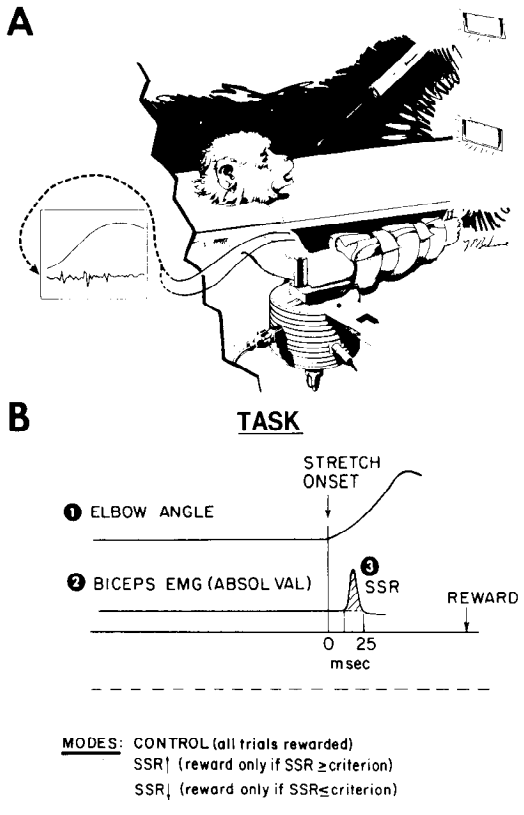

FIG. 1. Experimental design. *A*: animal performing task. Implanted arm rests in a cast attached to torque motor shaft. Elbow angle is monitored by rotary variable differential transformer below motor. Chronically implanted fine-wire electrodes, which exit at upper forearm, monitor EMG. Dim upper light 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↓ mode, reward occurs only if SSR amplitude, *3*, is above or below a preset value (reproduced from Ref. 16).

proached 100%, and incentive remained relatively constant.

After training to the control mode, monkeys normally completed 3,000–6,000 trials/day. 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 and was expressed in units of background EMG amplitude. As previously noted, all EMG measurements were absolute value. In addition, raw EMG and elbow angle were recorded on analog tape at regular intervals.

Data were gathered from each animal over 2–17 mo. Throughout this time, background EMG and the initial 30 ms of pulse-induced extension remained constant. For the first 10–60 days, the animal worked under the control mode to permit determination of naive SSR amplitude. Then seven of the eight animals were exposed to the SSR↑ or SSR↓ mode for 35–274 days. They were then exposed to the opposite mode (SSR↓ or SSR↑) for at least 35 days. Finally, four of these seven were reversed again, back to the SSR↑ or SSR↓ mode, for at least 40 days.

At various times during data collection, performance was interrupted for periods of 10–38 days. The connector plug was coated with silicon adhesive and buried under the skin. An animal spent all or most of a gap in its home cage. In some cases it spent 10–15 days of the gap performing the task with the other arm under the control mode. In these cases the original arm was free and entirely at the animal's disposal, much as in the home cage. It is important to note that at the end of a gap the animal always resumed performance under the same mode (control, SSR↑, or SSR↓) it was operating under prior to the gap.

Animal well-being and food and water consumption were meticulously monitored (16). All animals remained healthy and active throughout the study.

RESULTS

On first exposure to the SSR↑ or SSR↓ mode following the control-mode exposure, each monkey changed SSR amplitude appropriately over the succeeding weeks, without change in background EMG or the initial 30 ms of pulse-induced extension.[2] Following mode reversal and reexposure, SSR amplitude also changed appropriately. As previously reported in detail (15, 16, 18), the SSR↑ mode ultimately produced increases to 150% or more of immediate preexposure amplitude, while the SSR↓ mode ultimately produced decreases to ~50% of immediate preexposure amplitude.

As noted above, animals typically completed 3,000–6,000 trials/day throughout data collection. However, on return to performance

[2] The concurrent behavior of antagonist and synergist muscles has been described in detail (17). In brief, background EMG in these muscles did not change. Synergist SSR amplitude changed in the same direction as biceps SSR amplitude, but to a lesser degree.

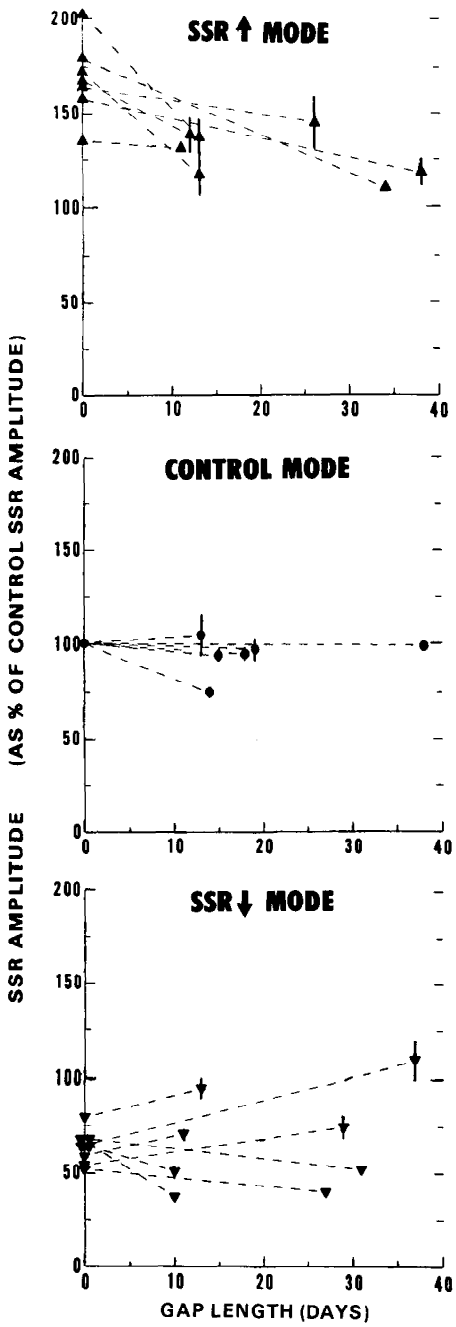

FIG. 2. Average SSR amplitude for the last 10 pregap days (*left-hand point* of each *pair*) and the first 5 postgap days (*right-hand point* of each *pair*) (±SEM) for all gaps under each mode. For the control mode gaps (6 from 4 animals), values are in percent of average SSR amplitude for 10 days immediately prior to the gap. For the SSR↑ gaps (6 from 4 animals) and SSR↓ gaps (8 from 6 animals) values are in percent of SSR amplitude immediately prior to that mode exposure (i.e., before the mode-induced adaptive change). The horizontal coordinate of the *right-hand point* is gap length. Control-mode gaps have little effect on SSR amplitude. SSR↑ mode gaps appear to cause progressive decay of adaptive SSR increase. SSR↓ mode gaps have varied effects, but consistent decay of adaptive SSR decrease is not apparent. See text for full discussion.

following a gap, animals sometimes began slowly, working only a relatively small number of trials in the first 1–2 days. In addition, partial retraining, to elbow angle and/or background EMG level, was sometimes necessary. Such retraining took from several hours to, at most, 2 days. In order to analyze the data from this immediate postgap period, we adopted two rules. First, any day with <300 trials was counted as an additional gap day. Second, any day with either 300–999 trials and/or average background EMG more than 20% different from pregap average background EMG was counted as a performance day for which the data were not interpretable. Usually we did not need to apply these rules. When they were applied they typically affected only the first 1 or 2 postgap days.

The eight animals were exposed to a total of 21 gaps of 10–38 days each under the control, SSR↑, or SSR↓ mode. For all the SSR↑ or SSR↓ mode gaps in this group, current mode exposure had been maintained for at least 5 wk prior to the gap, so that adaptive change was largely completed (18), and pregap SSR amplitude was at least 20% above (SSR↑) or below (SSR↓) SSR amplitude immediately prior to that mode exposure.

These data are presented in Fig. 2, illustrated in Fig. 3, and summarized in Table 1. In Fig. 2, each gap is represented by a pair of connected points. Control mode gaps (6 from 4 animals) are shown in the middle graph, SSR↑ gaps (7 from 4 animals) in the top graph, and SSR↓ gaps (8 from 6 animals) in the bottom graph. For each gap, the left-hand point shows SSR amplitude for the 10 days prior to the gap, and the right-hand point shows SSR amplitude for the 5 days immediately following the gap (±SEM). For the control-mode gaps, values are in percent of average SSR amplitude for the 10 days immediately prior to the gap. For SSR↑ and SSR↓ mode gaps, values are in percent of average SSR amplitude for the 10 days immediately prior to that mode exposure. The horizontal coordinate is gap length.

As Fig. 2 and Table 1 show, long gaps under the control mode had little effect on average SSR amplitude for the first 5 postgap days. Postgap amplitude is slightly less than pregap

amplitude, though the difference is not significant by correlated t test (t = 1.46, df = 5, $P > 0.1$). Under the SSR↑ mode, gaps are associated with significant degradation of the adaptive SSR increase produced by SSR↑ exposure. Pregap amplitudes average 168% of control, while postgap amplitudes average 128%. This drop is significant (t = 4.25, df = 6, $P < 0.01$). Furthermore, though the data are limited, they suggest that this loss of adaptive increase is correlated with gap length. Calculating for each gap the percent of adaptive increase lost per gap day and averaging the seven values gives a half-life estimate of 17 days. Figure 3*A* shows average postpulse EMG and elbow extension for 3 representative days from one animal exposed to a 13-day gap under the SSR↑ mode. The solid trace is a control day. The dashed trace is a day following prolonged SSR↑ exposure and immediately prior to the gap. The dotted trace is an early postgap day. Note that postgap SSR amplitude is smaller than pregap amplitude but still larger than original control amplitude.

In contrast to the SSR↑ gaps, gaps during SSR↓ exposure had no consistent effect on SSR adaptive change. The pregap amplitude of 63.6% of control is not significantly different from the postgap value of 66.4% (t = 0.32, df = 7, $P > 0.1$). Of the eight gaps, four show moderate decrease in amplitude, and three show moderate increase. Only one shows a marked increase (i.e., clear loss of adaptive change). Figure 3*B* shows average postpulse EMG and elbow extension for 3 representative days from one animal exposed to a 27-day gap under the SSR↓ mode. The solid trace is a control day. The dashed trace is a day following prolonged SSR↓ exposure and immediately prior to the gap. The dotted trace is an early postgap day. SSR decrease clearly persists over the gap.

Figure 2 and Table 1 are simply based on

TABLE 1. *Average pregap and postgap SSR amplitudes*

Mode	Pregap SSR Amp	Postgap SSR Amp
SSR↑	168.0 (±10.2)	128.0 (±5.3)
Control	100.0	93.8 (±4.6)
SSR↓	63.6 (±3.3)	66.4 (±9.9)

Average pregap and postgap SSR amplitudes (±SEM) under each mode in percent of immediate pregap SSR amplitude (for control mode gaps) or percent of immediate premode SSR amplitude (for SSR↑ and SSR↓ gaps).

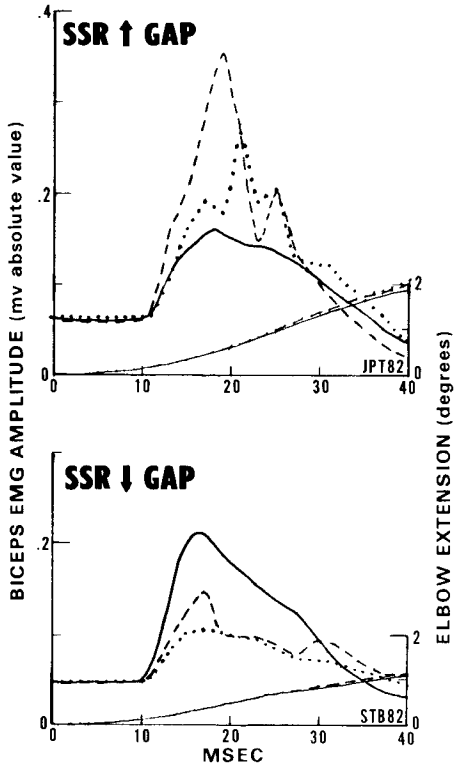

FIG. 3. *A:* average postpulse EMG and elbow extension for 3 representative days from an animal exposed to a 13-day gap under the SSR↑ mode. The *solid* trace is a control day. The *dashed* trace is a day following prolonged SSR↑ exposure and immediately prior to the gap. The *dotted* trace is an early postgap day. The initial 15–20 ms of pulse-induced elbow extension, the stimulus eliciting the SSR, is constant. The gap is associated with considerable loss of the SSR increase produced by SSR↑ exposure. *B:* analogous data from an animal exposed to a 27-day gap under the SSR↓ mode. There is no loss of the SSR decrease produced by SSR↓ exposure. The postgap SSR (*dotted*) is in fact slightly smaller than the pregap SSR (*dashed*).

SSR amplitude for the first 5 postgap days. They would not reveal very rapid postgap adaptive change. To rule out such change and detect any nonspecific gap effects, we evaluated SSR amplitude over these first days in detail. Figure 4 shows average daily SSR amplitudes (±SEM) for all gaps for the first 16 postgap days under each of the three modes. The horizontal dashed lines indicate average pregap amplitudes. Linear regression lines based on the first 10 postgap days are shown for each mode's data and are extended as dashed lines beyond 10 days. SSR amplitude is somewhat reduced after control-mode gaps and recovers gradually over the initial postgap days. The SSR↑ data show a comparable but somewhat

steeper rise. In contrast, the SSR↓ data remain stable over the initial postgap days. Thus, gaps appear to have a nonspecific effect on SSR amplitude. Amplitude is modestly depressed immediately afterward and recovers over days. The more rapid rise for the SSR↑ data is presumably due to superimposition of slow, phase II, SSR↑ adaptive change, whereas the lack of increase under the SSR↓ mode is probably due to slow, phase II, SSR↓ adaptive change, which cancels out the nonspecific postgap rise (see DISCUSSION).

As a further check on the possibility of very rapid postgap adaptive change, we evaluated each animal's first 1,000 postgap trials to the extent permitted by the 3-h data summaries. Figure 5, based on all available data, shows average SSR amplitude (±SEM) for each 100 trials for 6 SSR↑ gaps, 4 control gaps, and 5 SSR↓ gaps. [The gaps included in this figure were those that involved no retraining to background EMG, so that the results of the first 1,000 trials were interpretable (see above).] There is no evidence for rapid postgap adaptive change. (Since animals usually completed 3,000–6,000 trials/day, these first 1,000 trials cover only a small initial segment of the data in Fig. 4, and thus the nonspecific postgap increases seen in Fig. 4 are not apparent in Fig. 5.) In sum, Figs. 4 and 5 indicate that the Fig. 2 and Table 1 presentations of gap effects on adaptive change are accurate, though the presentations may have slightly underestimated the preservation of SSR↑ change and slightly overestimated the preservation of SSR↓ change.

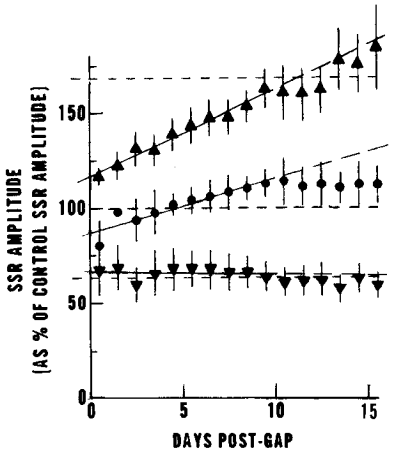

FIG. 4. Average daily SSR amplitudes (±SEM) for all gaps for the first 16 postgap days under each of the 3 modes. Linear regression lines are based on the first 10 days of postgap data. An immediate postgap decrease and gradual rise are apparent in the control mode and SSR↑ mode data. The rise is more rapid for the SSR↑ data. A rise is not apparent in the SSR↓ mode data. See text for full discussion.

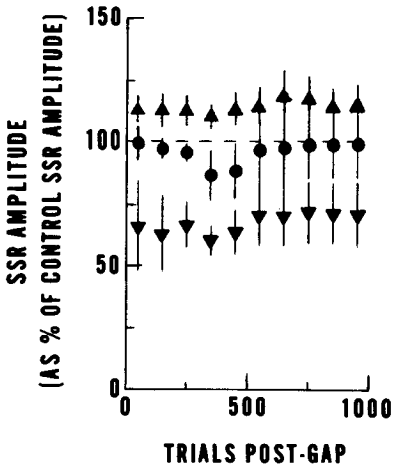

FIG. 5. Average SSR amplitude (±SEM) for each 100 of the first 1,000 postgap trials for all gaps for which proper background EMG allowed use of the very early postgap data (see RESULTS). The values are comprised of data from 6 SSR↑ gaps, 4 control gaps, and 5 SSR↓ gaps. There is no evidence for rapid adaptive change in these first postgap trials.

DISCUSSION

Previous studies (15, 16) demonstrated that SSR amplitude change occurs slowly over weeks and months, during initial development as well as during reversal and redevelopment. Detailed analysis (18, 19) of this slow course showed that it contained two components: nearly immediate phase I change, which occurs in the first 6 h, and very slow phase II change, which continues indefinitely and is responsible for at least 80–90% of the final change. As discussed previously (18), phase I appears to be an operantly conditioned phenomenon. It is presumably due to altered activity in one or more descending spinal cord pathways impinging on the segmental arc of the SSR (1). Because pulse-onset time is unpredictable and the SSR occurs before any other possible response, this change in descending activity must of necessity be tonic; it must be present day after day for a large part of the 5–7 h/day the animal spends on the task (18). The most probable interpretation (18) of phase II appears to be that it represents

persistent structural and/or biochemical alteration gradually produced by the chronic presence of the tonic descending activity responsible for phase I. Such alteration could be occurring on segmental and/or suprasegmental levels. If, as related laboratory and clinical evidence suggests (16), this persistent alteration occurs at the segmental level, it should constitute an accessible substrate of memory.

The present results are consistent with the presence of a persistent alteration, since they demonstrate that SSR adaptive change persists for substantial periods without continued task performance. Adaptive SSR increase produced by the SSR↑ mode declines slowly over weeks with a half-life of ~17 days. Adaptive SSR decrease produced by the SSR↓ mode shows no uniform decline. There is no evidence of rapid SSR retraining in the first postgap days under either the SSR↑ or SSR↓ mode. Instead, initial postgap days appear to show nonspecific initial depression and subsequent gradual recovery of SSR amplitude. The postgap rise is accentuated under the SSR↑ mode, presumably due to resumption of slow, phase II, SSR↑ change, and masked under the SSR↓ mode, presumably due to resumption of slow, phase II, SSR↓ change.

Immediate postgap behavior

The postgap rise is greater under the SSR↑ mode than under the control mode and is absent under the SSR↓ mode. As noted above, we ascribe this difference to postgap resumption of slow, phase II, adaptive change, which accentuates the rise under the SSR↑ mode and cancels it out under the SSR↓ mode. We can evaluate this interpretation by comparing the difference in slope between the SSR↑ and SSR↓ regressions in Fig. 4 to the phase II rates previously calculated (18). If the SSR↑ and SSR↓ slopes of Fig. 4 are each expressed in terms of average SSR amplitude over the first 10 postgap days, the amplitudes diverge at a rate of 3.5%/day. This closely matches a divergence of 3.4%/day (calculated in the same way) ascribable to phase II over the initial 10 days of mode exposure (18) and thus supports our interpretation of the postgap behavior seen in Fig. 4.

The apparent modest depression and subsequent rise in SSR amplitude in the first postgap days may be due to the more varied use of the extremity during the gap. For example, the greater range of elbow movement prevalent during the gap might induce γ-motoneuron-mediated reduction in spindle stretch sensitivity so that the spindle can provide suitable information throughout the full range of movement (8). Such reduction in spindle sensitivity could account for the reduction in SSR amplitude in the first days following task resumption, and an increase in spindle sensitivity with task resumption could explain the subsequent rise. Change in muscle bulk or mechanical state might also have a role, though the lack of significant change in the initial 30 ms of pulse-induced extension makes marked alteration in such features very unlikely.

Other factors that might affect persistence

The 21 gaps differed in other potentially important factors besides mode and length. The duration of SSR↑ or SSR↓ mode exposure prior to gap onset, while always at least 35 days, varied considerably. Previous mode exposure histories differed: For some SSR↑ or SSR↓ gaps there had been previous exposure to the opposite mode, as well as to the control mode. Some gaps were entirely spent in home cages, while portions of others were spent working under the control mode with the other arm. Within the significant limits imposed by our relatively limited body of data, we could detect no marked effects of these factors. This is perhaps not surprising. The major portion of adaptive change is established by 35 days (18), and the final effects of a given mode exposure are largely dependent on its starting point, not on the starting point of previous mode exposures. The primary arm's activity while the other is performing the task is not strikingly different from its activity in the home cage. In all cases the other arm was exposed to the control mode, and in any event, preliminary data suggest that adaptive change is confined to the arm working under the SSR↑ or SSR↓ mode. Nevertheless, the data are limited, and these factors, as well as others, may have effects on persistence that are not apparent here.

SSR↑ persistence versus SSR↓ persistence

In this study SSR↑ adaptive change slowly disappears without continued performance, whereas SSR↓ adaptive change usually persists. SSR↓ change also develops and redevelops more rapidly than does SSR↑ change (18). These differences are particularly interesting

in relation to other long-term changes in segmental reflex function, those associated with suprasegmental damage, normal development, and prolonged athletic training.

A major function of suprasegmental control is suppression of segmental reflexes. Loss or impairment of such control allows hyperreflexia. Increased spinal stretch reflexes typically accompany both lesions of motor cortex and subjacent structures and lesions of spinal cord descending pathways (4, 7, 10, 13). Conversely, over the first years of life segmental reflexes decline in amplitude and distribution as they are brought under greater suprasegmental control. This process may fail to occur in the presence of perinatal suprasegmental abnormalities (11, 12). Similarly, certain prolonged athletic training, directed at producing extremely precise control of limb movements, is associated with further suppression of segmental reflexes (5).

Thus, evidence of several kinds suggests that the SSR↑ mode requires a reduction in suprasegmental control, while the SSR↓ mode requires an increase. Viewed in this light, adaptive SSR increase is an atypical or abnormal event, the relinquishing of higher level control over segmental function, whereas adaptive SSR decrease is comparable to processes occurring with normal development and motor training. This difference may help account for the greater speed and persistence of adaptive SSR decrease.

ACKNOWLEDGMENTS

We thank Ms. Ellen Vander Schaaf for aid in data analysis.

This work was supported in part by grants from the United Cerebral Palsy Research and Educational Foundation (R-322-82&84) and the National Institutes of Health (NS-22189).

Received 1 March 1985; accepted in final form 4 September 1985.

REFERENCES

1. BALDISSERA, F., HULTBORN, H., AND ILLERT, M. Integration in spinal neuronal systems. In: *Handbook of Physiology. The Nervous System. Motor Control,* edited by J. M. Brookhart and V. B. Mountcastle. Bethesda, MD: Am. Physiol. Soc., 1981, sect. 1, vol. II, pt. 1, chapt. 12, p. 509–595.
2. BASMAJIAN, J. V. *Muscles Alive.* Baltimore, MD: Williams & Wilkins, 1978, p. 32–36.
3. BURKE, D., GANDEVIA, S.C., AND MCKEON, B. Monosynaptic and oligosynaptic contributions to human ankle jerk and H reflex. *J. Neurophysiol.* 52: 435–448, 1984.
4. DELWAIDE, P. J. Contribution of human reflex studies to the understanding and management of the pyramidal syndrome. In: *Electromyography in CNS Disorders: Central EMG,* edited by B. T. Shahani. Boston, MA: Butterworth, 1984, p. 77–109.
5. GOODE, D. J. AND VAN HOEVEN, J. Loss of patellar and Achilles tendon reflexes in classical ballet dancers. *Arch. Neurol.* 39: 323, 1982.
6. HENNEMAN, E. AND MENDELL, L. M. Functional organization of motoneuron pool and its inputs. In: *Handbook of Physiology. The Nervous System. Motor Control,* edited by J. M. Brookhart and V. B. Mountcastle. Bethesda, MD: Am. Physiol. Soc., 1981, sect. 1, vol. II, pt. 1, chapt. 11, 423–507.
7. LANCE, J. W. Pyramidal and extrapyramidal disorders. In: *Electromyography in CNS Disorders: Central EMG,* edited by B. T. Shahani. Boston, MA: Butterworth, 1984, p. 1–18.
8. LOEB, G. E. The control and responses of mammalian muscle spindles during normally executed motor tasks. *Exercise Sport Sci. Rev.* 12:157–203, 1984.
9. MATTHEWS, P. B. C. *Mammalian Muscle Receptors and Their Central Actions.* Baltimore, MD: Williams & Wilkins, 1972, p. 319–409.
10. MOUNTCASTLE, V. B. Effects of spinal cord transection. In: *Medical Physiology,* edited by V. B. Mountcastle. St. Louis, MO: Mosby, 1980, vol. I, p. 781–786.
11. MYKLEBUST, B. M., GOTTLIEB, G. L., AND AGARWAL, G. C. Stretch reflexes of the normal human infant. *Dev. Med. Child Neurol.* In press.
12. MYKLEBUST, B. M., GOTTLIEB, G. L., PENN, R. L., AND AGARWAL, G. C. Reciprocal excitation of antagonistic muscles as a differentiating feature in spasticity. *Ann. Neurol.* 12: 367–374, 1982.
13. SAX, D. S. AND JOHNSON, T. L. Spinal reflex activity in man: measurement in relation to spasticity. In: *Spasticity: Disordered Motor Control,* edited by R. G. Feldman, R. R. Young, and W. P. Koella. Chicago, IL: Year Book, 1980, p. 301–313.
14. WOLPAW, J. R. Reflexes capable of change: models for the study of memory. *Federation Proc.* 41: 2146, 1982.
15. WOLPAW, J. R. Adaptive plasticity in the primate spinal stretch reflex: reversal and re-development. *Brain Res.* 278: 299–304, 1983.
16. WOLPAW, J. R., BRAITMAN, D. J., AND SEEGAL, R. F. Adaptive plasticity in primate spinal stretch reflex: initial development. *J. Neurophysiol.* 50: 1296–1311, 1983.
17. WOLPAW, J. R., SEEGAL, R. F., AND O'KEEFE, J. A. Adaptive plasticity in primate spinal stretch reflex: behavior of synergist and antagonist muscles. *J. Neurophysiol.* 50: 1312–1319, 1983.
18. WOLPAW, J. R. AND O'KEEFE, J. A. Adaptive plasticity in the primate spinal stretch reflex: evidence for a two-phase process. *J. Neurosci.* 4: 2718–2724, 1984.
19. WOLPAW, J. R., O'KEEFE, J. A., KIEFFER, V. A., AND SANDERS, M. G. Reduced day-to-day variation accompanies adaptive plasticity in the primate spinal stretch reflex. *Neurosci. Lett.* 54: 165–171, 1985.