Probable Corticospinal Tract Control of Spinal Cord Plasticity in the Rat

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Chen, Xiang Yang and Jonathan R. Wolpaw. Probable corticospinal tract control of spinal cord plasticity in the rat. J Neurophysiol 87: 645–652, 2002; 10.1152/jn.00391.2001. Descending activity from the brain shapes spinal cord reflex function throughout life, yet the mechanisms responsible for this spinal cord plasticity are poorly understood. Operant conditioning of the H-reflex, the electrical analogue of the spinal stretch reflex, is a simple model for investigating these mechanisms. An earlier study in the Sprague-Dawley rat showed that acquisition of an operantly conditioned decrease in the soleus H-reflex is not prevented by mid-thoracic transection of the ipsilateral lateral column (LC), which contains the rubrospinal, reticulospinal, and vestibulospinal tracts, and is prevented by transection of the dorsal column, which contains the main corticospinal tract (CST) and the dorsal column ascending tract (DA). The present study explored the effects of CST or DA transection on acquisition of an H-reflex decrease, and the effects of LC, CST, or DA transection on maintenance of an established decrease. CST transection prior to conditioning prevented acquisition of H-reflex decrease, while DA transection did not do so. CST transection after H-reflex decrease had been acquired led to gradual loss of the decrease over 10 days, and resulted in an H-reflex that was significantly larger than the original, naive H-reflex. In contrast, LC or DA transection after H-reflex decrease had been acquired did not affect maintenance of the decrease. These results, in combination with the earlier study, strongly imply that in the rat the corticospinal tract (CST) is essential for acquisition and maintenance of operantly conditioned decrease in the H-reflex and that other major spinal cord pathways are not essential. This previously unrecognized aspect of CST function gives insight into the processes underlying acquisition and maintenance of motor skills and could lead to novel methods for inducing, guiding, and assessing recovery of function after spinal cord injury.

INTRODUCTION

The brain exerts both short-term and long-term control over the spinal cord. The short-term impact of cortical activation on the spinal cord has been recognized since 1870, when Fritsch and Hitzig stimulated the cortex of a dog and produced movement, and many subsequent studies have explored the roles of the cortex and other brain areas in exciting and inhibiting spinal motoneurons (Fritsch and Hitzig 1960; Kandel et al. 2000; Porter and Lemon 1993). However, the long-term effects of the brain on the spinal cord and the mechanisms through which it shapes spinal cord reflex patterns so that they support effective motor control remain poorly understood. It is clear that descending activity from the brain gradually changes the spinal cord during development, after supraspinal trauma, and during skill acquisition (reviewed in Wolpaw and Tennissen 2001); yet the pathways and the processes through which this activity induces and maintains spinal cord plasticity are not known. New possibilities for restoring function after spinal cord injury have drawn attention to the mechanisms by which the brain gradually shapes spinal cord reflexes so that they function properly during movement (Bregman 1998; Fawcett 1998; Ramer et al. 2000; Tuszynski and Kordower 1999). Understanding these mechanisms could lead to novel methods for inducing, guiding, and assessing recovery after injury.

Operant conditioning of the spinal stretch reflex (SSR), or its electrical analogue the H-reflex, is a simple laboratory model for studying the brain’s induction and maintenance of spinal cord plasticity (Wolpaw 1997). The SSR (or “tendon jerk”) is the initial response to sudden muscle stretch and is the simplest behavior of the vertebrate CNS (Brown 1984; Henneman and Mendell 1981; Magladery et al. 1951; Matthews 1972). It is mediated largely by a monosynaptic pathway consisting of the Ia afferent neuron from the muscle spindle, its synapse on the alpha motoneuron in the spinal cord, and the motoneuron itself. Monkeys, humans, and rats can increase or decrease SSR or H-reflex amplitude in response to a reward contingency (Chen and Wolpaw 1995; Evatt et al. 1989; Wolf et al. 1995; Wolpaw 1987; Wolpaw et al. 1983). Reflex increase (i.e., up-training) or decrease (i.e., down-training) occurs gradually over days and weeks and is accompanied by plasticity in spinal cord motoneurons, in the synaptic terminals on them, and probably in spinal interneurons as well (Carp and Wolpaw 1994, 1995; Carp et al. 2001; Feng-Chen and Wolpaw 1996; Wolpaw 1997).

This spinal cord plasticity appears comparable to that induced by the brain during normal development early in life, during skill acquisition later in life, and in response to spinal cord trauma (reviewed in Wolpaw 1997; Wolpaw and Tennissen 2001). That it depends for its initial production on pathways that connect the brain to the spinal cord is indicated by studies showing that spinal cord contusion impairs H-reflex training (Chen et al. 1996, 1999). Furthermore, destruction of the dorsal column in the rat, which contains the main corticospinal tract (CST) and the dorsal column ascending tract (DA), prevents down-training, while destruction of the ipsilateral...
lateral column (LC), which contains the rubrospinal, vestibulospinal, and reticulospinal tracts and several ascending tracts (Tracey 1995), does not do so (Chen and Wolpaw 1997). However, it is not known whether down-training depends on the CST, the DA, or both; and it is not known whether the CST, DA, and/or pathways in the LC are essential for the long-term maintenance of a smaller H-reflex once it has been acquired.

To determine whether the CST and/or the DA is essential for the acquisition of operantly conditioned decrease in the H-reflex, we transected the CST or DA and then attempted to down-train the H-reflex. To determine whether the CST, DA, or pathways in the LC are essential for the maintenance of a conditioned decrease in the H-reflex, we down-trained the H-reflex first, and then transected the CST, DA, or LC and observed whether the conditioned decrease in the H-reflex persisted or disappeared.

**METHODS**

Subjects were female Sprague-Dawley rats weighing 200–300 g at the beginning of study. All procedures satisfied the “Guide for the Care and Use of Laboratory Animals” of the Institute of Laboratory Animal Resources, Commission on Life Sciences, National Research Council (National Academy Press, Washington, D.C. 1996) and had been reviewed and approved by the Institutional Animal Care and Use Committee of the Wadsworth Center. The protocols for H-reflex conditioning and spinal cord pathway transection are described in detail elsewhere and summarized here (Chen and Wolpaw 1995, 1997; Chen et al. 2001; Wolpaw and Herchenroeder 1990).

Each rat was implanted under general anesthesia with chronic stimulating and recording electrodes in the right leg. To elicit the H-reflex, a nerve cuff was placed on the right posterior tibial nerve just above the triceps surae branches. To record soleus electromyographic (EMG) activity, fine-wire electrodes were placed in the right soleus muscle. The Teflon-coated wires from the nerve cuff and the muscle passed subcutaneously to a connector plug mounted on the skull.

Throughout the data collection period, each animal lived in a standard rat cage with a flexible cable attached to the skull plug. The cable, which allowed the animal to move freely about the cage, carried the wires from the electrodes to an electronic swivel above the cage and from there to an EMG amplifier and a nerve-cuff stimulation unit. All animals had free access to water and food, except that during H-reflex down-training they obtained food mainly as described below. A computer system continuously monitored soleus EMG and controlled the nerve-cuff stimulus. If the absolute value of background EMG remained within a defined range for a randomly varying 2.3- to 2.7-s period, a stimulus pulse (usually 0.5 ms in duration) was delivered by the nerve cuff. Pulse amplitude was continuously adjusted so that it remained just above M-response threshold. Background EMG, M-response amplitude, and stimuli per day remained stable throughout data collection. Under the control mode, the computer simply monitored the absolute value of soleus EMG for 50 ms following the stimulus and determined H-reflex amplitude. Under the down-training mode, it gave a food reward 200 ms after nerve stimulation if EMG amplitude in the H-reflex interval (typically 5.5–9.0 ms after stimulation) was below a criterion value.

For spinal cord pathway transection, the rat was anesthetized, the cord was exposed with a T9–T10 laminectomy, and transection was performed by electrocautery. The cauterizer was activated in brief pulses to minimize thermal damage to adjacent tissue. Each rat received one of three different transections: lateral column (LC rats), which includes rubrospinal, vestibulospinal, and reticulospinal tracts; main corticospinal tract (CST rats), located in the base of the dorsal column; or dorsal column ascending tract (DA rats), located dorsal to the main CST in the dorsal column (Chung et al. 1987; Cliffer and Giesler 1989; Holstege and Kuyper 1987; Patterson et al. 1989, 1990; Paxinos and Watson 1986; Smith and Bennett 1987; Tracey 1995; Zemlan et al. 1978, 1979). For LC rats, the lateral half of the right side of the spinal cord was transected. LC transection was ipsilateral to avoid the considerable disability likely to be associated with a bilateral LC transection (which would have destroyed about 2/3 of the white matter), and because, at the thoracic level, the major descending tracts in the LC (i.e., rubrospinal, vestibulospinal, and reticulospinal tracts) are mainly or exclusively ipsilateral to the leg they serve (Tracey 1995). For CST rats, the cauterizer was mounted in a micromanipulator, and the tip was positioned 1.0 mm left of the midline of the dorsal surface of the spinal cord, pointed medially at an angle of 45° from vertical, and advanced 1.7 mm so that it transected the CST bilaterally. For DA rats, transection extended 0.4 mm to either side of the midline and 0.7 mm into the spinal cord from the dorsal surface, and thus cut the DA bilaterally. After transection, the site was rinsed with normal saline and covered with Durafilm to minimize connective tissue adhesions to the dura, and the muscle and skin were sutured in layers. Care in the days immediately after transection included analgesia, antibiotics, bladder expression, and food supplementation as previously described in detail (Chen and Wolpaw 1997; Chen et al. 2001). Bladder function returned within several days, and locomotion returned to normal or nearly normal by 10 days.

At the end of study, each rat was killed by an overdose of pentobarbital sodium and perfused, the spinal cord was removed, and blocks encompassing the transection were embedded in paraffin. Transverse 10- to 20-μm-thick serial sections were cut and stained with Luxol fast blue. Camera lucida drawings were made at a magnification of 50. Remaining white matter was identified at a magnification of 200 by the presence of normal Luxol fast blue staining. The tracings were enlarged and then digitized, and the areas of specific pathways remaining at the epicenter of the transection were determined. Figure 1A shows T8–T9 transverse sections and corresponding camera lucida drawings from a normal rat, an LC rat, a CST rat, and a DA rat. For LC, the area of right LC remaining was measured as percent of the left LC. For CST or DA, the area remaining was measured as percent of the area of that structure 2–5 mm rostral to the rostral limit of the lesion.

Figure 1B shows the protocol of the acquisition study. An earlier study (Chen and Wolpaw 1997) had shown that LC transection does not prevent acquisition of an H-reflex decrease, while transection of the entire dorsal column does prevent it. To determine which of the dorsal column pathways, the CST or the DA, is needed for acquisition, we transected the CST or the DA and collected H-reflex data first under the control mode and then under the down-training mode. We then compared H-reflex amplitude at the end of down-training to control H-reflex amplitude to determine whether CST and/or DA transection prevented the decrease in the H-reflex that occurs in most normal rats exposed to the down-training mode.

Figure 1C shows the protocol for the maintenance study. To determine which pathways are essential for the maintenance of H-reflex decrease once it has been acquired, we collected H-reflex data under the control mode, down-trained the H-reflex over 50 days, transected LC, CST, or DA, and continued down-training for another 50 days. We then compared the final H-reflex amplitude to H-reflex amplitude just prior to transection to determine whether LC, CST, and/or DA transection abolished the decrease in the H-reflex that had been produced by down-training. We also compared the final H-reflex amplitude to H-reflex amplitude just prior to transection to evaluate more fully the effects of LC, CST, or DA transection.
RESULTS

Histology

In the 12 LC rats, 20 ± 22% (mean ± SD) of the right LC, all of the right CST, and 91 ± 22% of the right DA remained. In the 10 CST rats, 6 ± 15% of the right CST (and 3 ± 6% of the left CST) remained. While CST rats retained all of the right LC, most showed some loss of the right DA, with 58 ± 32% remaining. In the 11 DA rats, 18 ± 24% of the right DA, 99 ± 5% of the right CST, and all of the right LC remained. In CST and DA rats, the left DA and/or LC also showed variable damage. Because CST, DA, and the major descending LC tracts are ipsilateral at T8–T9, because comparable CST lesions do not affect H-reflex amplitude beyond the first 1–2 days in naive (i.e., untrained) rats (Chen et al. 2001), and because the extent of left LC or DA loss in CST rats did not correlate with final H-reflex amplitude (P > 0.3), this contralateral collateral damage does not account for the effects of CST transection on down-training of the H-reflex that are described below. Similarly, the variable collateral damage to adjacent mid-thoracic gray matter should not have affected the lumbar spinal cord, which mediates the soleus H-reflex, or its supraspinal connections. Additional data concerning the possible impact of collateral damage to ipsilateral pathways are presented below.

Acquisition study

Figure 2 summarizes the results of down-training in rats with CST or DA transection and includes for comparison earlier results from normal rats and from rats with LC transection (Carp et al. 2001; Chen and Wolpaw 1995–1997; unpublished data). The four groups differed significantly (P < 0.01 by ANOVA), and this difference was due to the CST rats. Down-training was similarly effective in normal, LC, and DA rats: final H-reflex amplitudes were 65 ± 3% (mean ± SE), 71 ± 8%, and 66 ± 6% of initial value, for normal, LC, and DA rats, respectively, and the final values for LC and DA rats did not differ significantly from each other, as shown in Figure 2.
differ from those of normal rats ($P > 0.5$ for each by Newman-Keuls test). In normal, LC, and DA rats, the H-reflex decrease was clearly significant ($P < 0.01$ by paired *t*-test for each group). In contrast, CST rats did not decrease the H-reflex in response to down-training: final H-reflex amplitudes averaged $103 \pm 6\%$ of control ($P > 0.6$ vs. control-mode values by paired *t*-test), and were clearly different from the final amplitudes for normal, LC, or DA rats ($P < 0.01$, $< 0.05$, or $< 0.05$, respectively, by Newman-Keuls test).

Because the major goal of this study was to compare the effects of CST and DA transections, the critical comparison is between CST and DA rats, and the possible role of ipsilateral collateral damage (i.e., to right DA in CST rats and to right CST in DA rats) must be considered. Table 1 shows for all CST and DA rats the percentages of right CST and DA remaining and the final H-reflex amplitudes at the end of down-training. Right CST destruction was complete in four of the five CST rats, and right DA destruction was complete in four of the six DA rats. All of the CST rats, including the two that retained nearly all (87 and 81%) of the right DA, failed to achieve successful down-training [i.e., a decrease of $\pm 20\%$ (Wolpaw et al. 1993)]. In contrast, five of the six DA rats showed successful down-training, and the only one that did not was also the only one that had any CST damage. In sum, the results presented in Fig. 2 and Table 1 strongly imply that the CST is essential for down-training of the H-reflex, and that the LC and DA are not essential.

**Maintenance study**

Figure 3 (top) summarizes the results obtained from 15 rats that were down-trained and then subjected to LC, CST, or DA transection (with 5 rats in each group). For the first 50 days of down-training, the H-reflex fell as expected (Chen and Wolpaw 1995), and averaged $55 \pm 4\%$ (mean $\pm$ SE) of control for days $41–50$, just prior to transection. It did not differ significantly among the three groups (i.e., $55 \pm 11\%$ for the LC rats, $60 \pm 7\%$ for the CST rats, $51 \pm 4\%$ for the DA rats; $P > 0.7$ by ANOVA). LC or DA transection had no detectable effect on the conditioned decrease: H-reflex amplitude remained the same or continued to drop slowly. For LC and DA rats, H-reflex amplitudes for days $91–100$, at the end of the second 50 days of down-training, averaged $53 \pm 17\%$ and $40 \pm 8\%$, respectively, and were not significantly different from those at the end of the first 50 days ($P > 0.05$ by paired *t*-test for both LC and DA rats). In contrast, after CST transection, H-reflex amplitude rose over about 10 days to above its original, control-mode amplitude, and remained there. H-reflex values for days $91–100$, at the end of the second 50 days of down-training, averaged $125 \pm 8\%$, and were significantly higher than those at the end of the first 50 days ($P < 0.01$), and also significantly higher than the control-mode values ($P < 0.04$). CST transection abolished the H-reflex decrease that had been acquired under the down-training mode.

As for the acquisition data, the most critical comparison for the maintenance data is between CST and DA rats, and the possible role of ipsilateral collateral damage (i.e., to right DA in CST rats and to right CST in DA rats) must be considered. Table 2 shows for all CST and DA rats the percentages of right CST and DA remaining, H-reflex amplitude just prior to transection, and final H-reflex amplitude. In the five CST rats, right CST destruction was complete or nearly complete, and the right DA remaining ranged from 100 to 0%. All five CST rats lost H-reflex down-training after the transection, including the two in which all or nearly all of the right DA remained. In the five DA rats, 1–64% of the right DA, and all of the right CST

![Figure 3](http://jn.physiology.org/)

**FIG. 3.** Results of maintenance study. Average H-reflex amplitudes ($\pm$SE) for each 5-day period for the 1st 50 days of down-training for all 15 rats (●) and for the next 50 days for the LC (▲), CST (●), and DA (▼) groups (5 rats in each group). LC or DA transection did not affect the H-reflex decrease achieved by down-training, while CST transection eliminated it over a period of about 10 days. The expansion shows average daily H-reflex amplitudes ($\pm$SE) for each group for the 2 days before and the 12 days after transection. As detailed in the text, it shows that all 3 transections produced a transient nonspecific increase in the 1st posttransection day, and that CST transection also produced gradual loss of the conditioned H-reflex decrease over about 10 days and a 25% increase that was still present 50 days later.

**Table 1.** Right CST and DA remaining and final H-reflex amplitude (% of control-mode value) for all CST and DA rats in the acquisition study

<table>
<thead>
<tr>
<th></th>
<th>CST Remaining</th>
<th>DA Remaining</th>
<th>Final H-Reflex</th>
</tr>
</thead>
<tbody>
<tr>
<td>CST rats</td>
<td>0</td>
<td>87</td>
<td>90</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>18</td>
<td>118</td>
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<tr>
<td></td>
<td>0</td>
<td>51</td>
<td>91</td>
</tr>
<tr>
<td></td>
<td>0</td>
<td>44</td>
<td>98</td>
</tr>
<tr>
<td></td>
<td>46</td>
<td>81</td>
<td>118</td>
</tr>
<tr>
<td>DA rats</td>
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</tr>
<tr>
<td></td>
<td>100</td>
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<td>62</td>
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<td>65</td>
</tr>
<tr>
<td></td>
<td>100</td>
<td>30</td>
<td>42</td>
</tr>
</tbody>
</table>

Values are percentages. CST, corticospinal tract; DA, dorsal column ascending tract.
remained. All five DA rats retained down-training after the transection, including the two in which only 1 or 2% of the right DA remained. In each of the five DA rats, the final H-reflex was smaller than the pre-transection H-reflex. Thus the results presented in Fig. 3 and Table 2 strongly imply that the CST is essential for the maintenance of down-training, and that the LC and DA are not essential.

Figure 3 (bottom) is a more detailed view of the days immediately before and after the pathway transections. All three transections appeared to increase the H-reflex in the first day (P < 0.01, <0.03, and <0.07 for LC, CST, and DA, respectively, for day 51 vs. days 49–50). This brief increase was seen also in naive (i.e., untrained) rats after these pathway transections (Chen et al. 2001), and was probably a nonspecific effect of the surgery and/or the accompanying general anesthesia. This transient effect disappeared by the second day, and from then on H-reflex amplitude in LC and DA rats was comparable to that just prior to transection. In contrast, CST rats gradually lost the H-reflex decrease produced by down-training. Over 10 days, H-reflex amplitude rose to 25% above its original control-mode value (linear trend P < 0.01) and remained there for the remainder of data collection. At the same time, the fact that the transient increase in the first day after transection was greater for CST rats than for LC or DA rats (and that H-reflex amplitude for CST rats was still substantially elevated on the 2nd day) suggested that the 25% rise responsible for the persistent increase occurred immediately after transection.

Figure 4 shows average poststimulus EMG from an LC rat, a CST rat, and a DA rat for representative days before down-training, at the end of down-training and just before transection, and after transection and continued down-training. With exposure to down-training, all three rats decreased the H-reflex over 50 days. LC or DA transection did not affect this decrease; it was still present 7 wk later at the end of data collection. In contrast, CST transection produced loss of the H-reflex decrease and led to an H-reflex that was somewhat larger than the original, control-mode H-reflex. Background EMG and M response remained the same throughout data collection.

**DISCUSSION**

In rats not exposed to the H-reflex down-training protocol, CST transection has no effect on H-reflex amplitude beyond the first 1–2 days, and LC or DA transection produces a modest long-term increase, not a decrease, in H-reflex amplitude (Chen et al. 2001). Thus the abolition of both acquisition and maintenance of a conditioned H-reflex decrease by CST transection implies that CST transection interferes with the processes

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**TABLE 2.** Right CST and DA remaining, and pre-transection and final H-reflex amplitudes (% of control-mode value), for all CST and DA rats in the maintenance study

<table>
<thead>
<tr>
<th></th>
<th>CST Remaining</th>
<th>DA Remaining</th>
<th>Pre-Transection H-Reflex</th>
<th>Final H-Reflex</th>
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<td>CST rats</td>
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<td>44</td>
<td>20</td>
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</table>

Values are percentages. For abbreviations, see Table 1.
Physiological studies in both monkeys and rats indicate that down-training of the H-reflex changes the motoneuron (Carp and Wolpaw 1994; Carp et al. 2001), and anatomical studies reveal plasticity in several populations of synaptic terminals on the motoneuron (Feng-Chen and Wolpaw 1996). After successful down-training, motoneuron firing threshold is more positive, possibly due to a change in sodium channel activation voltage, and this threshold change could largely account for the smaller H-reflex (Carp and Wolpaw 1994; Halter et al. 1995). The likelihood that neuronal plasticity underlies H-reflex down-training contrasts with the traditional emphasis on synaptic plasticity as the basis of learning (Martin et al. 2000). At the same time, it is consistent with other recent evidence (Spitzer 1999) that synaptic input can change neuronal firing properties by modifying voltage-gated ion channels. Up-training of the H-reflex appears to depend on different mechanisms (Carp and Wolpaw 1995; Wolpaw and Chen 2001). Initial data suggest that acquisition of up-training depends on the CST while maintenance does not, and that neither depends on the LC or DA (Chen et al. 2000).

Figure 3 (bottom) suggests that several different processes affect H-reflex amplitude in down-trained rats subjected to CST transection. While a variety of different interpretations might explain the data, the interpretation described in RESULTS and discussed here appears to be the simplest and most reasonable. It seems that the spinal cord plasticity responsible for the H-reflex decrease erodes over about 10 days once the CST activity that induces and maintains it is removed. This interpretation is compatible with the fact that conditioned decrease in the primate H-reflex is still evident 3 days after complete spinal cord transection (Wolpaw and Lee 1989). At the same time, it contrasts with the finding in intact monkeys that conditioned decrease in the SSR persists essentially unchanged for ≥4 wk after down-training stops (Wolpaw et al. 1986). The contrast suggests that the adaptive change in CST activity induced by down-training is not confined to the training periods, but is rather a tonic change that persists between training periods, and even persists for weeks after training terminates. This implication is consistent with the course of SSR down-training in humans, which progresses at a rate similar to that found in monkeys and rats even though the humans have only short periods of training each day (Evatt et al. 1989; Wolf et al. 1995).

Figure 3 indicates that down-training also produces an unexpected change in spinal cord function, a 25% increase in H-reflex amplitude over its initial, control-mode amplitude that is obvious only after CST transection permits erosion of the plasticity underlying the H-reflex decrease. This change is unexpected because CST transection in naive rats does not produce a comparable lasting increase (Chen et al. 2001). This 25% increase, which persists unchanged to the end of data collection, might reflect additional, more persistent spinal cord plasticity or a persistent change in activity in the remaining descending pathways. It may be related to the plasticity in the contralateral spinal cord that occurs with H-reflex down-training in primates and is evident only after spinal cord transection removes descending control (Wolpaw and Lee 1989). It is further evidence that even the simplest behavioral changes involve plasticity at multiple spinal and/or supraspinal sites (Carrier et al. 1997; Cohen et al. 1997; Garcia et al. 1999; Lieb and Frost 1997; Lisberger 1998; Thompson et al. 1997;
Whalen and Pearson 1997; Wolpaw 1997; Wolpaw and Lee 1989). Such complex plasticity appears to be both necessary (to maintain performance of other behaviors) and inevitable (due to the ubiquity of activity-dependent plasticity in the CNS) (Wolpaw 1997; Wolpaw and Tennissen 2001). As noted in RESULTS, the data suggest that this 25% increase is present immediately after transection. This possibility might be evaluated by testing the effects of reversible short-term inactivation (e.g., Garcia and Mauk 1998) of sensorimotor cortex.

Both laboratory and clinical studies suggest that spinal cord plasticity comparable to that underlying H-reflex conditioning is important for effective motor control in normal life. Spinally mediated muscle stretch reflexes and flexion withdrawal reflexes, which are poorly focused and often inappropriate in newborn infants, become precisely and appropriately focused during early life, and this development depends on normal descending control from the brain (Levinsson et al. 1999; Myklebust et al. 1982, 1986; O’Sullivan et al. 1991; Wolpaw 1997; Wolpaw and Tennissen 2001). Later in life, muscle stretch reflexes and H-reflexes change gradually during skill acquisition (Koceja et al. 1991; Meyer-Lohmann et al. 1986; Nielsen et al. 1993; Wolpaw 1997; Wolpaw and Tennissen 2001). Disorders that disrupt descending activity, such as spinal cord injury or stroke, also produce gradual long-term changes in spinal cord reflexes, and these changes contribute to spasticity and other disabling problems (Hiersemenzel et al. 2000; Kuhn 1950; Riddoch 1917; Ronthal 1998).

The role of the CST in shaping spinal cord reflexes, demonstrated by the present data, may have significant practical implications. The capacity for H-reflex down-training could be a sensitive and specific measure of CST function after spinal cord injury and of the success of interventions intended to promote CST regeneration. Furthermore, exploration of the mechanisms underlying the long-term impact of the CST on the spinal cord could help define the requirements for restoring function to a newly regenerated spinal cord, and could lead to new methods for inducing and guiding that restoration. Finally, this newly recognized CST function gives insight into the acquisition of those motor skills that are acquired through prolonged practice (Wolpaw and Tennissen 2001). Such skills are likely to involve spinal cord plasticity, and thus may not be adequately explained by plasticity that occurs in cortex, cerebellum, or other supraspinal areas. The long-term influence that the CST exerts over spinal cord function may contribute to the acquisition and maintenance of a wide variety of normal behaviors.

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