Dorsal Column But Not Lateral Column Transection Prevents Down-Conditioning of H Reflex in Rats

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Chen, Xiang Yang and Jonathan R. Wolpaw. Dorsal column but not lateral column transection prevents down-conditioning of H reflex in rats. *J. Neurophysiol.* 78: 1730–1734, 1997. Operant conditioning of the H reflex, the electrical analogue of the spinal stretch reflex, in freely moving rats is a relatively simple model for studying long-term supraspinal control over spinal cord function. Motivated by food reward, rats can gradually increase or decrease the soleus H reflex. This study is the first effort to determine which spinal cord pathways convey the descending influence from supraspinal structures that changes the H reflex. In anesthetized Sprague-Dawley rats, the entire dorsal column (DC), which includes the main corticospinal tract, or the right lateral column (LC) was transected by electrocautery. Animals recovered quickly and the minimal transient effects of transection on the right soleus H reflex disappeared within 16 days. Beginning at least 18 days after transection, 12 rats were exposed to the HRdown-conditioning mode, in which reward was given when the H reflex of the right soleus muscle was below a criterion value. In seven LC rats exposed to the HRdown mode, the H reflex fell to 71 ± 8% (mean ± SE) of its initial value. In six of the seven, conditioning was successful (i.e., decrease to ≤80%). These results were comparable with those previously obtained from normal rats. In contrast, in five DC rats exposed to the HRdown mode, the H reflex at the end of exposure was 106 ± 12% of its initial value. In none of these rats was HRdown-conditioning successful. DC rats differed significantly from normal and LC rats in both final H reflex values and number successful. In five DC and three LC rats that continued under control conditions over 30–78 days, the H reflex at the end of the period was 98 ± 4% and 100 ± 8%, respectively, of its initial value, indicating that DC or LC transection itself did not lead to gradual increase or decrease in the H reflex. The results indicate that the DC, containing the main corticospinal tract, is essential for HRdown-conditioning, whereas the ipsilateral LC, containing the main rubrospinal, vestibulospinal, and reticulospinal tracts, is not essential. Combined with the known muscular specificity of conditioning, these results suggest that the main corticospinal tract is essential for HRdown-conditioning. The DC ascending tract might also be necessary. The respective roles of the DC descending and ascending tracts, and transection effects on HRup-conditioning and on the maintenance of both HRup- and HRdown-conditioning after they have occurred, remain to be defined.

INTRODUCTION

The spinal stretch reflex (SSR), the simplest behavior of the vertebrate CNS, is mediated by a wholly spinal and largely monosynaptic pathway consisting of the primary afferent neuron, the α-motoneuron, and the synapse between them. Operant conditioning of the SSR or its electrical analogue, the H reflex, has been demonstrated in monkeys (Wolpaw 1987; Wolpaw and Lee 1989), humans (Evatt et al. 1989; Wolf and Segal 1990, 1996), and rats (Chen and Wolpaw 1995a,b, 1996). Motivated by a paradigm in which reward depends on reflex amplitude, both primates and rats can gradually increase or decrease the SSR or the H reflex. The conditioning paradigm appears to induce a change in descending influence that modifies the spinal cord and changes the reflex (Carp and Wolpaw 1994; Feng-Chen and Wolpaw 1996; Wolpaw and Lee 1989).

Recent studies in rats show that contusion injuries to thoracic spinal cord impair operant conditioning of the soleus H reflex and that the degree of impairment is correlated with the size of the lesion (Chen et al. 1996). These results confirm the essential role of spinal cord pathways in H reflex conditioning. However, because contusions cause diffuse damage, they do not indicate which pathways are essential for conditioning.

This study is the first effort to define the roles of specific spinal cord pathways in H reflex conditioning. We investigate the effects on down-conditioning of transecting the dorsal column (DC), which in rats contains the main corticospinal tract, or the lateral column (LC), which contains the rubrospinal, vestibulospinal, and reticulospinal tracts (Holstege and Kuypers 1987; Kennedy 1990; Kuypers 1981; Tracey 1995). The results are quite clear, and with further exploration they should lead to greater understanding of long-term supraspinal control over spinal cord function and of the spinal reflex abnormalities that occur when injury impairs that control.

METHODS

Subjects were 18 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, DC 1996) and had been reviewed and approved by the Institutional Animal Care and Use Committee of the Wadsworth Center. The protocol for monitoring and conditioning the H reflex in freely moving rats has been described in detail elsewhere (Chen and Wolpaw 1994, 1995a,b, 1996) and is summarized here. The spinal cord lesion protocol is described fully.

Each rat was implanted under general anesthesia (ketamine HCl 80 mg/kg ip and xylazine 10 mg/kg ip) with chronic stimulating and recording electrodes in the right leg. To elicit the H reflex, a silicone rubber nerve cuff containing a pair of stainless steel multistranded fine-wire electrodes was placed on the right posterior tibial nerve just above the triceps surae branches. To record soleus electromyographic (EMG) activity, a pair of fine-wire electrodes with the final 0.5 cm stripped 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

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skull. Data collection started ≥10 days after implantation. During data collection, each animal lived in a standard rat cage with a 40-cm 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 to food, except that during H reflex conditioning they received food mainly by performing the task described below. Animal well-being was carefully checked several times each day, and body weight was measured weekly. Laboratory lights were dimmed from 2100 to 0600 h each day.

A computer system continuously monitored soleus EMG and controlled the nerve cuff stimulus. If the absolute value (i.e., equivalent to the full-wave rectified value) of background (i.e., ongoing) EMG remained within a defined range for a randomly varying 2.3- to 2.7-s period, a stimulus pulse (0.5 ms in duration for most animals) was delivered by the nerve cuff. Pulse amplitude was initially set just above M response threshold and then continuously and automatically adjusted to maintain M response amplitude unchanged throughout the weeks of data collection. Under the control mode, the computer simply measured the absolute value of soleus EMG for 50 ms following the stimulus. Under the HRdown-conditioning mode, the computer gave a reward (i.e., a 20-mg food pellet) 200 ms after nerve stimulation if EMG amplitude in the H reflex interval (5.5–9.0 ms after stimulation in a typical animal) was below a criterion value. In the course of its normal activity, the animal usually satisfied the background EMG requirement, and thus received nerve cuff stimulation, 2,500–8,000 times per day. H reflex amplitude was calculated as average EMG amplitude in the H reflex interval minus average background EMG amplitude and was expressed in units of average background EMG amplitude.

For transection of the DC (8 rats) or LC (10 rats), the animal was anesthetized as for electrode implantation and a one-vertebra dorsal laminectomy was performed at T_8 or T_9 with minimal disturbance of the dural envelope. The rat was placed in a stereotaxic frame and the cord was visualized under a dissection microscope. For DC rats, electrocautery was used to transect the DCs bilaterally (i.e., lesion extending 0.4 mm to either side of the midline and 1.1 mm into the cord). For LC rats, electrocautery was used to transect the right LC (i.e., the lateral 0.9 mm of the cord on the right side). DC transection was bilateral because we wanted to evaluate the importance to conditioning of all DC pathways. LC transection was ipsilateral because we wished to avoid, particularly in this initial study, the considerable disability likely to be associated with a bilateral LC lesion (which would have destroyed ~2/3 of the white matter), and because the major rubrospinal, vestibulospinal, and reticulospinal tracts are ipsilateral (Tracey 1995). During transection, the cautery was activated in brief pulses to minimize thermal damage to adjacent tissue. After transection, the site was rinsed with saline and covered with Durafilm to minimize connective tissue adhesions to the dura, and the muscle and skin were sutured in layers.

Immediately after transection, the rat was placed under a heating lamp and given an analgesic (Demerol, 0.2 mg im). Once awake, the rat received a second dose of analgesic and was returned to its cage and allowed to eat and drink freely. Until spontaneous voiding returned, the bladder was expressed at least three times daily and antibiotics [Gentocin (gentamicin sulfate) 0.25 mg im b.i.d. and Flo-Cillin (penicillin G benzathine and penicillin G procaine) 15,000 U im q.o.d.] and lactated Ringer solution (5 ml sc b.i.d.) were given. For the first 5 days after transection, the animal was given a soft mash of water-soaked rat chow with added vitamin C (~8 $mg \cdot kg^{-1} \cdot day^{-1}$ to keep urine acidic to prevent urinary tract infections). Body weight was measured daily and a high-calorie dietary supplement (Nutri-Cal; 2–4 ml/day po) was given until body weight regained its prelesion level. At least 10 g of apple were given each day from before transection until the end of the study.

In 12 rats (6 DC and 6 LC), electrode implantation preceded transection by at least 33 days, and control mode data were collected before transection from 11 rats. The other six rats (2 DC and 4 LC) were transected 16–17 days before implantation, and collection of control mode data began 25–71 days after transection. For all rats, posttransection control mode data were collected over periods of 11–78 days. Beginning 18–85 days after transection and after collection of control mode data, 12 rats (5 of the 8 DC rats and 7 of the 10 LC rats) were exposed to the HRdown mode for 50 days (except for 1 LC rat that lost the head plug after 33 days).

To determine the effect on H reflex amplitude of exposure to the HRdown mode, average H reflex amplitude for the final 10 days of HRdown exposure was calculated as percent of average H reflex amplitude for the final 10 days of control mode exposure. In addition, for those animals in which posttransection control mode data collection lasted ≥30 days, average H reflex amplitude for the final 10 days was calculated as percent of the average for the first 10 days. This analysis, along with comparison of posttransection control mode data with data collected before transection, assessed the effects of the DC and LC transections themselves on H reflex amplitude.

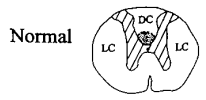
At the end of study, each rat was given an overdose of pentobarbital sodium (intraperitoneally) and perfused through the heart with saline followed by 4% paraformaldehyde (or 3% paraformaldehyde and 1% glutaraldehyde) in 0.1 M phosphate buffer (pH 7.3). The placement of the EMG electrodes and the nerve cuff and the integrity of the tibial nerve were verified, and the soleus muscles of both sides were removed and weighed. The spinal cord was removed and blocks encompassing the lesion were embedded in paraffin. Transverse sections (20 μm) were cut from the paraffin-embedded blocks and stained with Luxol fast blue (for myelinated fibers) and 0.1% cresyl violet (for Nissl substance). Sections encompassing the T_8–T_9 level of the lesion were assessed to determine the location and size of the lesion. For LC rats, the area of LC remaining was calculated as percent of the contralateral LC. (Nearly identical values were obtained when LC area was calculated as percent of the ipsilateral LC 2–5 mm rostral to the lesion.) The border between LC and the ventral column was defined according to Paxinos and Watson (1986). For DC rats, the area of DC remaining was calculated as percent of the DC 2–3 mm rostral to the lesion (which was comparable in area with the DC of normal rats).

RESULTS

Immediately after DC or LC transection, rats showed a transient hindlimb paralysis (both hindlimbs for DC rats and right only for LC rats) that abated rapidly over 1–3 days. For all rats, locomotion about the cage appeared normal or nearly normal within 4–10 days. Bladder function, absent immediately after injury, returned over 1–7 days. Although the time to return of bladder function was longer for DC rats (3.4 ± 2.2 days, mean ± SD) than for LC rats (2.3 ± 1.2 days), the difference was not statistically significant ($P = 0.2$, *t*-test). Body weight fell 2–13% in the 1st week after transection and regained its pretransection level in 2–5 wk. Weight increased from 299 ± 42 g at transection to 354 ± 58 g at perfusion. Soleus muscles weights (measured as % body weight) were symmetrical and did not differ significantly from normal.

Figure 1 shows camera lucida drawings of T_8–T_9 transverse sections from a normal rat and from the five DC rats

and seven LC rats exposed to the HRdown-conditioning mode and gives for the lesioned rats the percent of DC or right LC that remained the day posttransection when HRdown exposure began and the final H reflex amplitude after HRdown exposure. In DC rats, 96–100% of the targeted column was destroyed. In LC rats, 30–100% of the right LC was destroyed.



	DC/LC Remaining	HRdown Start Day	Final H-reflex
DC-1	0%	51	85%
DC-2	0%	23	99%
DC-3	0%	86	105%
DC-4	0%	31	153%
DC-5	4%	21	89%
LC-1	0%	42	49%
LC-2	0%	21	76%
LC-3	12%	33	77%
LC-4	12%	18	80%
LC-5	21%	51	64%
LC-6	50%	21	48%
LC-7	70%	35	105%

FIG. 1. Camera lucida drawings of transverse sections of T_8–T_9 spinal cord from a normal rat [with lateral column (LC) and dorsal column (DC) labeled and main corticospinal tract stippled] and from the 5 DC rats and 7 LC rats exposed to the HRdown-conditioning mode. In DC and LC rats, the section shown is at the lesion epicenter. Hatching: gray matter. Also shown for DC and LC rats are the percentage of the targeted structure remaining, the day postlesion when HRdown exposure began, and H reflex amplitude at the end of HRdown-conditioning (% of its initial value, i.e., its value for the 10 days immediately before HRdown-conditioning).

DC and LC transection had only modest short-term effects on H reflex amplitude, background EMG, and number of trials per day. In the 11 rats from which control mode data were collected before and after transection, all measures had returned to very near their pretransection control mode values by 16 days after transection. In four DC and two LC rats in which control mode data were collected before transection and as late as 30–85 days after transection, final control mode H reflex amplitudes were 106 ± 7% (mean ± SE) and 110 ± 0%, respectively, of their pretransection values. Most important in the present context (because the control mode data used to assess the effect of HRdown exposure were obtained after transection), prolonged posttransection control mode data collection gave no evidence for gradual effects of transection on H reflex amplitude. In five DC and three LC rats in which posttransection control mode data were collected over periods of 30–78 days after the transient effects of transection had disappeared, values for the final 10 days were 98 ± 4% (mean ± SE) and 100 ± 8%, respectively, of the values for the first 10 days. Over the whole period of data collection in all rats, background EMG and M response amplitude, which were controlled as indicated in the METHODS section, remained stable.

Figure 2 displays final H reflex amplitudes for the five DC rats and seven LC rats exposed to the HRdown mode and compares them with data from 14 normal rats similarly exposed (Chen and Wolpaw 1995a and subsequent data). Filled triangles indicate that HRdown-conditioning was successful (i.e., the H reflex decreased to ≤80% of its initial value) (Chen and Wolpaw 1995a; Wolpaw et al. 1993). The groups differed significantly ($P < 0.01$) according to analysis of variance. Pairwise comparisons were made with the use of the Newman-Keuls test, and, in regard to number successful, by the Fisher exact test. In the normal group, final H reflex amplitude averaged 68 ± 6% (mean ± SE), and 12 of 14 (86%) rats were successful. Results for the LC group were nearly identical: final H reflex amplitude averaged 71 ± 8% (mean ± SE, $P > 0.7$ vs. normal group) and six of seven (86%) were successful ($P > 0.9$ vs. normal group). In contrast, final H reflex amplitude in the DC group averaged 106 ± 12% (mean ± SE) and none of five (0%) rats was successful. The DC group differed from the normal and LC groups in both final value ($P < 0.01$ and $P < 0.05$, respectively) and number successful ($P < 0.005$ and $P < 0.02$, respectively).

No correlation was detected in DC or LC rats between H reflex amplitude at the end of HRdown exposure and time after transection when exposure began ($P > 0.8$ and $P > 0.7$, respectively). This was consistent with the spinal cord contusion data (Chen et al. 1996) and was additional evidence that the lesions themselves did not have long-term effects on H reflex amplitude. Furthermore, although the completeness of transection varied in the LC group, no correlation was detected in that group between final H reflex amplitude and tissue remaining ($P > 0.3$).

DISCUSSION

Exposure to the HRdown mode decreased the H reflex in LC animals and did not decrease it in DC animals. Neither

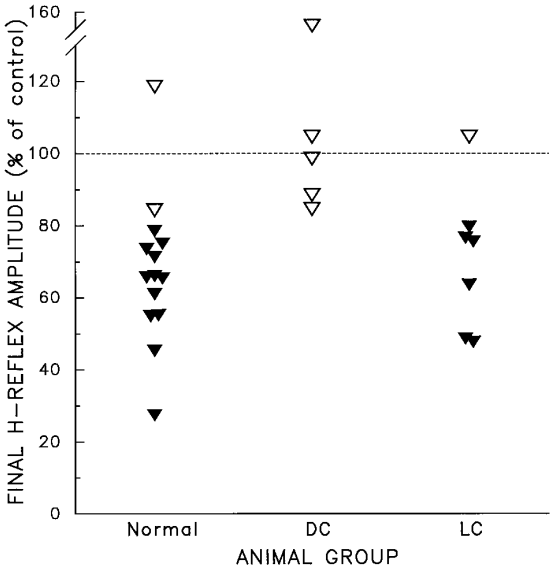


FIG. 2. Final H reflex values (% of initial values) for all normal, DC, and LC rats exposed to HRdown-conditioning. Filled triangles: HRdown-conditioning was successful (i.e., decrease to ≤80%). (Normal results are from Chen and Wolpaw 1995a and additional unpublished data.)

DC or LC transection alone had a noticeable persistent effect on H reflex amplitude. Under continued control mode exposure, H reflex amplitude several months after transection was comparable with that before transection and several weeks after transection. Thus the success of HRdown-conditioning in LC rats cannot be ascribed to lesion-induced H reflex decrease, and its failure in DC rats cannot be ascribed to lesion-induced H reflex increase. In addition, the success of HRdown-conditioning was not correlated with the amount of the ipsilateral LC destroyed, further suggesting that it is not essential for HRdown-conditioning. Indeed, the single LC rat in which HRdown-conditioning failed (*LC-7*, Fig. 1) had the smallest lesion. Although both DC and LC transections were often accompanied by some damage to adjacent thoracic gray matter and its associated propriospinal fibers, this additional damage should not have significantly affected function in the midlumbar spinal cord or impaired its communication with supraspinal structures. Thus the primary implication of these results seems clear: the DC is essential for HRdown-conditioning and the ipsilateral LC is not.

In the rat spinal cord, the major occupants of the DC are the main corticospinal tract and the sensory tract that ascends to the nuclei cuneatus and gracilis (Tracey 1995). Thus, on the basis of the present results, either one or both could be essential for HRdown-conditioning. Although the role of the ascending sensory tract is uncertain, several considerations suggest that the corticospinal tract is needed.

In primates, SSR and H reflex conditioning are relatively specific to the muscle being conditioned (Wolf et al. 1995; Wolpaw et al. 1983, 1989, 1993). Even when the reflexes of synergist muscles or the contralateral homonymous muscle are elicited at the same time throughout conditioning (so that their sensory fibers are also stimulated), mode-appropriate reflex change is greatest in or limited to the muscle that controls reward. Although the behavior of other muscles has not been studied during H reflex conditioning in the rat, conditioning in the rat is comparable with that in the primate in other respects (Chen and Wolpaw 1995a,b, 1996), so that a similar muscular specificity is probable. The corticospinal tract projects with high topographical specificity to both distal and proximal muscles (Kennedy 1990; Kuypers 1981; Porter and Lemon 1993) and thus could account for the highly focused nature of H reflex conditioning. Furthermore, it is believed to be especially important for learning new movements (Kennedy 1990; Kuypers 1981; Porter and Lemon 1993).

DC ascending fibers could not account for the muscular specificity of conditioning, because, as noted above, simultaneous stimulation of other muscles (or their nerves), which presumably produces comparable sensory input, does not lead to comparable change in their reflexes. Furthermore, the proprioceptive sensory input conveyed by these fibers is not needed to account for the operantly conditioned change in descending influence that changes the H reflex. Delivery of the food-pellet reward provides the sensory feedback necessary for guiding the change in descending influence.

The tentative conclusion that the corticospinal tract has an essential role in HRdown-conditioning is consistent with human data indicating that vascular lesions affecting motor cortex and related subcortical areas prevent down-conditioning of the SSR (Segal 1997). Nevertheless, definitive resolution of the respective roles of the DC descending and ascending tracts will require study of the effects of tract-specific transections (achieved, for example, by carefully placed electrolytic lesions).

Although the results suggest that the rubrospinal, vestibulospinal, and reticulospinal tracts are not essential for HRdown-conditioning, these tracts are not exclusively ipsilateral at midthoracic levels (Tracey 1995), so that the effects of bilateral LC lesions require evaluation. Also worthy of consideration, although probably of lesser importance because of the ipsilaterality of DC tracts, are potential differences between the effects of ipsilateral and bilateral DC lesions. Additional important issues remain. These include the following: whether transection of the ventral column (which contains the minor corticospinal tract) impairs conditioning; which tracts are essential for HRup-conditioning; and which tracts (if any) are essential for the long-term maintenance of HRup or HRdown-conditioning once it has occurred. Recent evidence that HRup and HRdown-conditioning have different spinal mechanisms (Carp and Wolpaw 1994, 1995) suggests that they might also depend on different tracts. The apparent importance of the rubrospinal tract, located in the dorsal LC, for already learned or automated movements (Kennedy 1990; Kuypers 1981) suggests that it may be important for the maintenance of H reflex conditioning. Although conditioned H reflex change can persist for days after spinal cord transection (Wolpaw and Lee 1989), its long-term maintenance (i.e., over wk and mo) is likely to require continued descending influence (Chen and Wolpaw 1996; Wolpaw et al. 1986).

Resolution of these issues should enhance understanding of H reflex conditioning specifically and of long-term supraspinal control of spinal cord function generally. This knowledge should help clarify the reflex abnormalities that occur with spinal cord injury and could provide a basis for the design of new therapeutic interventions.

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