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RESEARCH NOTE

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Operant conditioning of rat H-reflex: effects on mean latency and duration

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Abstract We are currently studying the mechanisms of operantly conditioned changes in the H-reflex in the rat. Primate data suggest that H-reflex decrease is due to a positive shift in motoneuron firing threshold and a small decrease in the monosynaptic excitatory postsynaptic potential (EPSP), and that increase might be due to change in group-I oligosynaptic (especially disynaptic) input. To further evaluate the possibility of conditioned change in oligosynaptic input, we compared the mean latency (i.e., the average latency of the entire H-reflex) and the duration of control (i.e., pre-conditioning) H-reflexes with those of H-reflexes after up-conditioning or down-conditioning. Up-conditioning was associated with small, statistically significant increases in H-reflex mean latency [+0.11±0.05 (±SE) ms] and duration (+0.32±0.16 ms). The mean latency of the H-reflex increase (i.e., the part added to the H-reflex by up-conditioning) was 0.28±0.14 (±SE) ms greater than that of the control H-reflex. Down-conditioning had no significant effect on mean latency or duration. While these results indicate that operant conditioning does not greatly change H-reflex mean latency or duration, the effects detected with up-conditioning are consistent with the hypothesis that decreased inhibition, or increased excitation, by homonymous and heteronymous group-I oligosynaptic input contributes to the H-reflex increase produced by up-conditioning. Several other mechanisms might also account for these small effects.

Keywords H-reflex · Conditioning · Spinal cord · Plasticity · Memory

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Introduction

Rats, monkeys, and humans can gradually increase or decrease the H-reflex and/or the spinal stretch reflex in response to an operant conditioning protocol in which reward is based on reflex amplitude (reviewed in Wolpaw 1997). Reflex change begins quickly and, then, continues to develop over days and weeks. It is associated with persistent functional and structural changes in the spinal cord itself. Intracellular recordings from motoneurons suggest that operantly conditioned H-reflex decrease (HRdown conditioning) in monkeys is due in large part to a positive shift in motoneuron firing threshold and a small decrease in the amplitude of the monosynaptic Ia-afferent excitatory postsynaptic potential (EPSP) in the motoneuron (Carp and Wolpaw 1994). HRdown conditioning in monkeys and rats is accompanied by a decrease in motoneuron axonal conduction velocity (Carp and Wolpaw 1994, Carp et al. 2000). In accord with this finding and with the lengthy peripheral pathway in the monkey, HRdown conditioning in the monkey is also associated with a commensurate increase in H-reflex latency. A modeling study supported the hypothesis that both the positive shift in motoneuron firing threshold and the decrease in conduction velocity result from a positive shift in the activation voltage of sodium channels in the motoneuron membrane (Halter et al. 1995).

On the other hand, intracellular motoneuron data from monkeys have not provided an explanation for the H-reflex increase that occurs with HRup conditioning (Carp and Wolpaw 1995). The presence of anesthesia might have prevented detection of conditioning-induced effects, such as a reduction in presynaptic inhibition at the Ia synapse. Nevertheless, the intracellular results, the likelihood that oligosynaptic (especially disynaptic) pathways can convey group-I homonymous and heteronymous inhibition to the motoneuron fast enough to affect the H-reflex (Baldissera et al. 1981; Jankowska 1992; Stephens and Yang 1996), and the evidence that HRup conditioning is associated with small-

er F terminals (i.e., inhibitory terminals) on the cell body and proximal dendrites of the motoneuron (Feng-Chen and Wolpaw 1996) suggest that conditioned H-reflex increase could be due to a decrease in the strength of group-I oligosynaptic pathways conveying inhibitory input to the motoneuron. A change in oligosynaptic excitation might also contribute to H-reflex change (Jankowska et al. 1981; Kirkwood and Sears 1982; McCrea et al. 1995; Angel et al. 1996; Stephens and Yang 1996).

If change in oligosynaptic inputs from group-I afferents does play a role in the H-reflex increase produced by HRup conditioning, it would not be expected to affect the latency of the beginning of the H-reflex (i.e., the onset latency), which presumably reflects the monosynaptic component. However, it might well increase the mean latency of the H-reflex, that is the average latency of all the electromyographic (EMG) activity that constitutes the H-reflex, because it would increase the latter part of the H-reflex more than the earlier part. Mean latency would increase because a decrease in oligosynaptic inhibition allowed underlying oligosynaptic excitation or the later part of monosynaptic excitation to excite the motoneuron and/or because oligosynaptic excitation increased. H-reflex duration might also increase. Change in H-reflex latency or duration with HRup conditioning was not apparent in the monkey. However, the relatively long latency and duration of the monkey triceps-surae H-reflex, combined with the limited temporal resolution of the measurement method, might well have obscured subtle changes. The rat soleus H-reflex has a shorter latency and a briefer duration than the monkey triceps-surae H-reflex, and the resolution of measurement is now better. Thus, the effect of a change in oligosynaptic input is more likely to be apparent. This study assessed the effects of HRup and HRdown conditioning on the mean latency and duration of the rat soleus H-reflex.

Materials and methods

Subjects

Subjects were 65 Sprague-Dawley rats (33 males and 32 females, initially weighing 203–646 g) with soleus H-reflexes in the normal range (i.e., 0.5–1.5× background EMG, as described below) prior to conditioning. They were implanted and conditioned as described below. 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 H-reflex conditioning protocol for rats is described in detail elsewhere (Chen and Wolpaw 1995) and summarized here. Calculation of the mean latency and the duration of the H-reflex before and after HRup or HRdown conditioning, and the mean latency of the H-reflex increase (by HRup conditioning) or decrease (by HRdown conditioning) is described fully.

Animal preparation and conditioning

Each rat was implanted under general anesthesia [i.e., intraperitoneal ketamine HCl (80 mg/kg) and xylazine (10 mg/kg) or pentobarbital (60 mg/kg)] with chronic stimulating and recording electrodes in the right hindlimb. To elicit the H-reflex, a silicone-rubber nerve cuff carrying a pair of stainless-steel multi-stranded fine-wire electrodes was placed around the right posterior tibial nerve just proximal to the triceps-surae branches. To record soleus EMG activity, a pair of fine-wire electrodes with the final 0.5 cm stripped were inserted in the right soleus muscle. The Teflon-coated wires from the nerve cuff and the muscle traveled subcutaneously to a connector plug on the skull. Data collection started at least 10 days later. During collection, the rat lived in a standard rat cage with a 40-cm flexible cable connected to the skull plug. The cable, which permitted the rat to move freely about the cage, conveyed 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. The rat had free access to water and food, except that during H-reflex conditioning it received food primarily by performing the task described below. Animal well-being was checked carefully several times each day, and body weight was measured each week. Laboratory lights were dimmed from 21:00 to 06:00 daily.

A computer system constantly 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 specified range for a randomly varying 2.3–2.7 s period, a stimulus pulse (usually 0.5 ms in duration) was delivered by the nerve cuff. Pulse amplitude was initially set just above the M-response threshold and, then, automatically and continuously adjusted to keep M-response amplitude unchanged over the weeks of data collection. Under the control mode, the computer simply digitized (at ≥2000 Hz) and stored the absolute value of the soleus EMG for 50 ms after nerve stimulation. Under the HRup or HRdown conditioning mode, it gave a food reward 200 ms after stimulation if the EMG amplitude in the H-reflex interval (typically 5.5–9.0 ms after stimulation) was greater than (HRup mode) or less than (HRdown mode) a criterion value. In the course of its normal activity, the rat satisfied the background EMG requirement, and thus received nerve-cuff stimulation, many times (2000–10 000/day, depending on the rat). Each rat was exposed to the control mode for at least 10 days and, then, to the HRup or HRdown mode for at least 50 days (except in a few rats in which loss of the head mount or malfunction of the implanted electrodes ended data collection after 30–49 days of HRup or HRdown exposure).

The computer provided a daily summary, which included number of trials, number of rewards, average background EMG amplitude immediately prior to nerve stimulation, and the course of average EMG amplitude for 50 ms after stimulation. H-reflex amplitude was defined as the average EMG amplitude in the H-reflex interval minus the average background EMG amplitude and was expressed in units of the average background EMG amplitude.

At the end of the study, each rat was given an overdose of sodium pentobarbital (i.p.) 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.

Data analysis

For each rat, the average H-reflex amplitude for the final 10 days of HRup or HRdown exposure was calculated as the percent of the average H-reflex amplitude for the final 10 days of control-mode exposure. This measure assessed the effect of the exposure to the HRup or HRdown mode on the H-reflex amplitude.

For each rat, the daily data for the final 10 days of control-mode exposure were averaged to give the average EMG amplitude immediately prior to nerve stimulation and for each 0.5-ms seg-

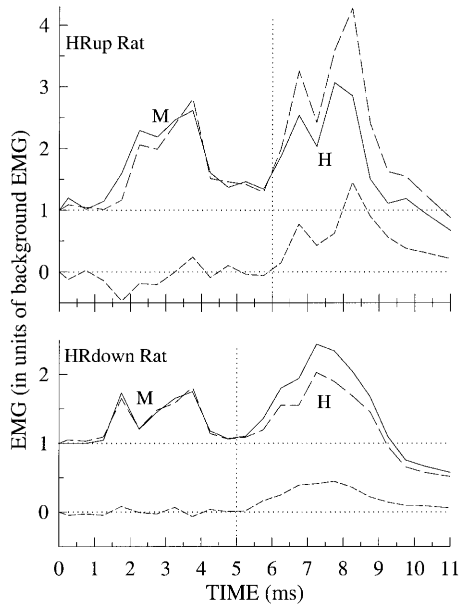

Fig. 1 Average control (*solid*), conditioned (*long dash*), and difference (*short dash*) traces for a rat exposed to the HRup conditioning mode (*HRup Rat*) and a rat exposed to the HRdown conditioning mode (*HRdown Rat*). In each rat, the M response (*M*) did not change with conditioning, while the H-reflex (*H*) underwent mode-appropriate change. The difference trace (conditioned trace minus control trace for the HRup rat and control trace minus conditioned trace for the HRdown rat) displays this change in isolation. The *vertical dotted line* marks the beginning of the first 0.5-ms segment in the H-reflex interval used for calculation of mean latency and duration by the equations shown in the text. (This interval always ended at 11 ms)

ment for the first 11 ms after the stimulus (i.e., a total of 22 segments). The daily data for the final 10 days of HRup or HRdown exposure were similarly averaged. These average control and conditioned data for the first 11 ms after the stimulus are subsequently referred to as control and conditioned traces.

In order to calculate, from each rat's control and conditioned traces, mean latencies for its control and conditioned H-reflexes and for the H-reflex increase (the part added by HRup conditioning) or the H-reflex decrease (the part subtracted by HRdown conditioning), the onset of the H-reflex was normally defined as the beginning of the first 0.5-ms segment after the segment with the lowest value between the M response and the H-reflex (e.g., Fig. 1). For each rat, this H-reflex onset was the same for control and conditioned traces. (Differences in this onset across rats were probably due mainly to differences in size and, thus, in peripheral nerve length.) Then, H-reflex mean latency was calculated from the EMG values of all the segments between the onset of the H-reflex and 11.0 ms that were >1 (i.e., that exceeded background EMG) by subtracting 1 from each value (to remove background EMG), multiplying the remainder by the latency of the middle of its segment, summing the products across all the included segments, and dividing the sum by the sum of the remainders for all the included segments. That is, if V_i was voltage at segment i, ML was H-reflex mean latency, and the H-reflex began at the beginning of segment B, ML was:

$$ML = \sum_{i=B,Vi>1}^{22} ((i/2) - 0.25)(V_i - 1) \Big/ \sum_{i=B,Vi>1}^{22} (V_i - 1)$$

(Thus, mean latency, or ML, was a weighted average of the latencies of the midpoints of the segments that contributed to the H-reflex, with the weights determined by the EMG amplitudes of the segments. It was essentially the center of gravity of the H-reflex.)

For each rat, this formula provided mean latencies for the control and conditioned H-reflexes (i.e., ML_{CN} and ML_{CD}, respectively).

Calculation for each HRup rat of ML_{INC}, the mean latency of the H-reflex increase, started from the fact that the conditioned H-reflex was the sum of the control H-reflex and the increase and, therefore, if A_{CN} and A_{CD} were the amplitudes of the control and conditioned H-reflexes:

$$ML_{CD} = \frac{(ML_{CN})(A_{CN}) + (ML_{INC})(A_{CD} - A_{CN})}{A_{CD}}$$

Solving for ML_{INC} gave:

$$ML_{INC} = \frac{(ML_{CN})(A_{CN}) - (ML_{CN})(A_{CN})}{A_{CD} - A_{CN}}$$

In an analogous fashion, for each HRdown rat, the mean latency of the H-reflex decrease (the part subtracted by HRdown conditioning) (ML_{DEC}) was calculated as:

$$ML_{DEC} = \frac{(ML_{CN})(A_{CN}) - (ML_{CD})(A_{CD})}{A_{CN} - A_{CD}}$$

For each rat's control and conditioned H-reflexes, duration was calculated as 0.5 ms times the number of segments that contributed to the calculation of mean latency, as described above.

The measure of H-reflex mean latency defined here was not significantly contaminated by the threshold M response, which usually peaked at 2–4 ms and was very small or absent by the beginning of the first H-reflex segment (e.g., Fig. 1). The selection of 11 ms as the end of the H-reflex interval confined the latency and duration measures to the expected H-reflex period. At the same time, because most control traces returned to or went below background EMG amplitude prior to 11 ms, this cutoff gave sufficient time to detect an increase in H-reflex duration. The data provided a sensitive assessment of the effects of HRup or HRdown conditioning because the mean latency was calculated from 6–10 0.5-ms segments, weighted according to the amplitudes of the H-reflex in the segments, because the average control and conditioned H-reflexes of each rat were each calculated from at least 20 000 single trials and because many rats were studied. [In a computer simulation (using H-reflexes of realistic shapes over appropriate ranges of variation in onset time, duration, and magnitude of amplitude change, and digitizing at 2000 Hz – i.e., the actual rate), the standard deviation of the measured mean latency about the true mean latency was 0.02 ms, or 20 µs, for one rat and 0.005 ms, or 5 µs, for the group averages. In a comparable simulation of the measurement of H-reflex duration, the standard deviation of the measured duration about the true duration was 0.19 ms for one rat and 0.04 ms for the group averages. These tests indicated that the measurement methods were able to resolve the effects reported below].

Results

H-reflex conditioning was successful [i.e., change ≥20% in the correct direction (Wolpaw et al. 1993; Chen and Wolpaw 1995)] in 22 of 29 rats (76%) exposed to the HRup mode and 26 of 36 rats (72%) exposed to the HRdown mode. In the 22 successful HRup rats, H-reflex amplitude rose to 174±63% (±SD) of its control mode value, and in the 26 successful HRdown rats it fell to 58±19%. In each of the 17 HRup or HRdown rats in which H-reflex conditioning was not successful, final H-reflex amplitude remained within 20% of its control-mode value, averaging 106±15% (±SD) in the seven unsuccessful HRup rats and 97±14% in the ten unsuccessful HRdown rats. In both HRup and HRdown groups, average background EMG amplitude and average M re-

Table 1 Average (±SD) mean latency and duration for control and conditioned H-reflexes of HRup and HRdown rats, and average mean latency for the H-reflex increase (*HRup* rats) and H-reflex decrease (*HRdown* rats). The *asterisks* indicate a significant ($P<0.05$, paired *t*-test) difference from the control H-reflex in the predicted direction

		Control H-reflex	Conditioned H-reflex	H-reflex increase or decrease
HRup rats (n=22)	Mean latency (ms)	7.49±0.61	7.60±0.61*	7.78±0.80*
	Duration (ms)	4.41±0.96	4.73±0.91*	–
HRdown rats (n=26)	Mean latency (ms)	7.53±0.54	7.47±0.67	7.57±0.66
	Duration (ms)	4.40±0.71	4.13±0.77	–

sponse amplitude remained stable (i.e., changed by <1%). Because the goal was to define the effects of conditioning on H-reflex latency and duration, the analysis presented here focused on the data from the 48 rats in which H-reflex conditioning was successful. Comparable analysis of the data of the 17 rats in which H-reflex conditioning was not successful, conducted to rule out nonspecific effects of the conditioning protocols, indicated that unsuccessful HRup or HRdown conditioning had no detectable effects on H-reflex mean latency or duration.

Figure 1 shows, for representative successful HRup and HRdown rats, control and conditioned traces and the difference between them. In both rats, the M responses of control and conditioned traces are nearly identical, while the H-reflexes show mode-appropriate differences. These differences, the H-reflex increase produced by HRup conditioning or the decrease produced by HRdown conditioning, are shown in isolation in their respective difference traces. [Because EMG measurement was absolute value, the number of peaks in the H-reflex is not meaningful. Two peaks (e.g., Fig. 1, top) might simply reflect the trial-to-trial stability in shape and onset time of a bipolar H-reflex].

Table 1 shows average (±SD) mean latency and duration of control and conditioned H-reflexes of HRup and HRdown rats and average mean latency of the H-reflex increase (HRup rats) or decrease (HRdown rats). For HRup and HRdown rats, we compared (by a paired *t*-test) the mean latencies and durations of the conditioned H-reflexes with those of the control H-reflexes. In addition, for HRup rats, we compared the mean latencies of the H-reflex increases with those of the control H-reflexes; and, for HRdown rats, we compared the mean latencies of the H-reflex decreases with those of the conditioned H-reflexes (i.e., the H-reflexes that remained after HRdown conditioning).

In HRup rats, the mean latency and duration of the conditioned H-reflexes were significantly greater (P=0.02 and 0.03, respectively) than those of the control H-reflexes. The increases, while significant, were modest: 0.11±0.05 ms (±SE) for mean latency and 0.32±0.16 ms for duration. As would be expected, the difference from the control was greater for the mean latency of the H-reflex increase itself: it averaged 0.28±0.14 ms more than the mean latency of the control H-reflex (P=0.03).

In HRdown rats, the mean latency of the conditioned H-reflex was only 0.07±0.07 ms (±SE) less than that of the control H-reflex and 0.11±0.15 ms less than that of the H-reflex decrease. Neither difference was significant (P>0.15 and 0.23, respectively). The duration of the conditioned H-reflex was 0.27±0.15 ms less than that of the control H-reflex, but this difference did not reach significance (P=0.07).

Discussion

The results show that, while operant conditioning of the rat H-reflex greatly changes H-reflex amplitude, it does not have major effects on mean latency or duration. This finding is consistent with the less-precise latency data from primates (e.g., Carp and Wolpaw 1994) and simple observation of the form of primate spinal stretch reflexes and H-reflexes before and after operant conditioning (e.g., Wolpaw et al. 1983; Wolpaw 1987). It also confirms initial observations of rat H-reflexes before and after conditioning (Chen and Wolpaw 1995). At the same time, the data show that HRup conditioning produces small, but significant increases in mean latency and in duration.

The significant increase in H-reflex mean latency with HRup conditioning implies that the mean latency of the H-reflex increase, that is, the part added to the H-reflex, averages 0.28 ms more than the mean latency of the control H-reflex. If the control H-reflex were exclusively monosynaptic and the increase exclusively disynaptic, the latency difference would be expected to be about 0.5 ms (i.e., one synaptic delay). Nevertheless, the 0.28-ms difference, and the accompanying increase in H-reflex duration, are consistent with a substantial disynaptic contribution to the H-reflex increase. As noted in the introduction, homonymous and heteronymous group-1 oligosynaptic inhibitory and excitatory inputs to the motoneuron have been described (Baldissera et al. 1981; Jankowska et al. 1981; Kirkwood and Sears 1982; Jankowska 1992; McCrea et al. 1995; Angel et al. 1996; Stephens and Yang 1996) and might well participate in the H-reflex (Burke et al. 1984; Fournier et al. 1986). H-reflex conditioning might change inhibition and/or excitation conveyed to the motoneuron by these pathways. The electron-microscopic evidence from monkeys for

less inhibitory input in HRup animals than in HRdown animals (Feng-Chen and Wolpaw 1996) suggests that the conditioning-induced change in oligosynaptic input could be reduced inhibition in HRup animals. These changes would increase motoneuron excitation by concurrent mono- and oligosynaptic excitatory input. On the other hand, other explanations for these effects are conceivable.

In monkeys, HRdown conditioning affects motoneuron axonal conduction velocity and H-reflex latency (Carp and Wolpaw 1994). However, because the peripheral pathway is much shorter in rats, the comparable conduction velocity changes (Carp et al. 2000) should have very small effects on H-reflex latency (i.e., probably ≤0.1 ms) (Birren and Wall 1956; Brunner et al. 1980; Sato et al. 1985; Chen et al. 1992). For the same reason, and because a large majority of rat soleus motor units are slow and have a limited range of conduction velocities, conditioning-induced changes in the population of soleus motoneurons recruited into the H-reflex (e.g., recruitment of motoneurons with higher conduction velocities with HRup conditioning) is also unlikely to produce substantial (i.e., >0.1 ms) changes in H-reflex latency (Ho et al. 1983; Gillespie et al. 1987; Taguchi et al. 1991; Walters and Constable 1993; Carp et al. 1999), and any change that did occur would presumably be a latency decrease rather than an increase. A more plausible possibility is a latency or duration change resulting from a change in the population of motor units contributing to the H-reflex. Conditioning-induced addition of stronger motor units might possibly affect H-reflex mean latency or duration by adding or subtracting motor units with wider EMG signatures. Finally, while changes in monosynaptic EPSP amplitude or motoneuron firing threshold should affect the number, but not the mean latency, of excited motoneurons, they might have some effect on timing and, combined with changes in motoneuron firing synchrony (e.g., less-perfect synchrony with the addition of motor units), could conceivably account for much of the latency and duration change seen with HRup conditioning.

In summary, while the results are consistent with the hypothesis that a decrease in short-latency oligosynaptic inhibition (or an increase in short-latency oligosynaptic excitation) of the motoneuron by homonymous and heteronymous group-Ia and/or -Ib afferents contributes to the H-reflex increase produced by exposure to the HRup conditioning mode, they are not a strong or definitive support for it. Because the observed latency and duration effects are modest, other explanations are also plausible. Current investigations of single motor-unit behavior during conditioning and of conditioning effects on conduction velocity and motor unit properties should provide further insight.

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