Spinal reciprocal inhibition in human locomotion

Aiko Kido, Naofumi Tanaka, and Richard B. Stein

1Centre for Neuroscience, University of Alberta, Edmonton, Alberta, Canada T6G 2S2;
2Department of Rehabilitation Medicine, Keio University School of Medicine, Tokyo 1608582, Japan;
and 3Department of Physiology, University of Alberta, Edmonton, Alberta, Canada T6G 2S2

MODULATION OF SPINAL INHIBITION has been studied in several motor tasks in humans (14, 17, 41, 42, 52, 56). In general, reciprocal inhibition is influenced by voluntary activation of antagonist muscles in various postures (e.g., lying, sitting, standing) in healthy humans, likely from a central origin (32, 40, 43, 48). The effect of antagonist muscle nerve stimulation on tonically active human single motoneurons was first examined by Ashby and colleagues (1, 3, 37). Reciprocal inhibition was observed as the period of reduced firing probability in a peristimulus time histogram of the target muscle, which appeared at the latency similar to that of the H reflex.

The costs of publication of this article were defrayed in part by the payment of page charges. The article must therefore be hereby marked "advertisement" in accordance with 18 U.S.C. Section 1734 solely to indicate this fact.

http://www.jap.org 8750-7587/04 $5.00 Copyright © 2004 the American Physiological Society
to running is generally clear and strong (12, 13, 25, but see also Refs. 24, 27, 53, and 54).

METHODOLOGICAL ISSUES

In contrast, the task-dependent modulation of reciprocal inhibition has not been studied to a similar extent. Some of the reasons are methodological. Although many studies have analyzed inhibition of the H reflex (11, 14, 17, 32, 36, 39, 41, 42, 48, 52, 56, 57), H-reflex inhibition is problematic during walking. The H reflex changes greatly during the step cycle and often is not seen at all during some phases (12, 36, 51). Inhibition and excitation are also very dependent on the size of the H reflex (15). Also, it might be difficult to ensure that the tibial nerve stimulus was unchanged, as well as the common peroneal stimulus throughout the dynamic movement (9). Therefore, in the present study, inhibition was measured as a decrease in the ongoing EMG activity, not as a decrease in the H reflex.

Modulation of reciprocal inhibition was studied in relation to three different tasks (i.e., standing, walking, and running) at several different speeds in healthy adults. The question asked was whether reciprocal inhibition is modulated between different tasks. First, the effect of stimulus intensity on the amount of inhibition was examined to determine a proper stimulation for studying a task dependence. In other words, we reexamined whether a weaker stimulation (i.e., 1.1 × MT) is better to observe a task dependence, as argued by Petersen et al. (49). Then, for the first time, reciprocal inhibition was investigated during running and compared with the inhibition during standing and walking. We found that the inhibition decreased substantially with increasing speed but showed a task-dependence, which was only significant in some comparisons.

MATERIALS AND METHODS

General procedure. Sixteen neurologically normal subjects aged from 25 to 61 yr participated in this study. All subjects gave informed consent for the purposes and procedures of the experiments, as approved by the Human Ethics Committee of the University of Alberta. EMG was recorded from the TA and soleus muscles with surface self-adhesive Ag-AgCl electrodes (Kendall LTP, Chicopee, MA). At the beginning of each experiment, the maximum tonic voluntary contraction (MVC) of each muscle was determined as the maximum rectified EMG level. During standing, the subject was asked to voluntarily control a target muscle EMG level for either plantar flexion or dorsiflexion to match a preset level on an oscilloscope (see EMG recordings and electrical stimulation). Then, reciprocal inhibition of the ankle extensors and flexors was elicited by stimulating the CP or the tibial nerve, respectively, while the subject was standing upright, walking, or running on a treadmill.

For 14 subjects, reciprocal inhibition of the soleus EMG activity was examined by CP nerve stimulation. First, the effect of stimulus intensity on the amount of inhibition in the soleus EMG activity was investigated. Under fixed tonic contraction (≈15% MVC) of the soleus, the CP nerve stimulus intensity was varied from the TA threshold to the maximum M wave. Four trials were recorded at each stimulus intensity. Collected data were immediately analyzed by a custom-written program in MATLAB (Mathworks, Natick, MA) to determine the MT of CP nerve stimulation for further experimental procedures. Then, the effect of soleus contraction level on the amount of inhibition was tested at a variety of soleus tonic contraction levels by using 1.5 × MT of the CP nerve stimulation, which produced clear inhibition in all subjects. Similarly, inhibition of soleus EMG activity elicited by the CP nerve stimulation was studied during walking. Ten of 14 subjects were asked to walk on a treadmill at a comfortable speed (≈4 km/h) for 4 min, while the CP nerve stimulation (1.5 × MT) was applied at all parts of the step cycle with random intervals (1.6–2.7 s). The same protocols were used for the tibial nerve stimulation and the inhibition of TA voluntary activity. For seven subjects, a stimulus intensity of 1.1 × MT was also used for both the CP and tibial nerve stimulation to examine the effect of stimulus intensity on the task-dependent modulation of reciprocal inhibition. From the comparison of results between two stimulus intensities (see RESULTS and Fig. 3), 1.5 × MT of stimulation was chosen for the rest of the study. Subsequently, in eight subjects inhibition of the ongoing EMG activity was investigated by stimulating either the CP nerve or the tibial nerve during standing, walking at either 3 or 6 km/h, and jogging or running at either 6 or 9 km/h. Amplitudes of M waves were monitored throughout each experiment to maintain a steady level of stimulation.

EMG recordings and electrical stimulation. The soleus and TA EMG signals were obtained by using surface self-adhesive Ag-AgCl electrodes (Kendall LTP). EMG recording electrodes were placed 2–3 cm below the gastrocnemius in line with the Achilles tendon for the soleus, and over the motor point for the TA, with –2-cm interelectrode spacing. The signals were amplified, high-pass filtered at 10 Hz, low-pass filtered at 1 kHz, and recorded with an 8-channel data acquisition system (Analog-Digital Interface, Union City, CA) at a sample rate of 5 kHz. EMG signals were also rectified, low-pass filtered at 3 Hz, and sent to an oscilloscope so that subjects could monitor their EMG activity levels during experiments. During standing EMG and nerve stimulus signals were recorded for 300 ms, including a prestimulus period of 40 ms, in response to each test stimulus pulse. During walking, heel contact was detected by a force-sensitive resistor (Interlink, Camarillo, CA) inserted between a subject’s shoe and foot. Heel contact was used to define the beginning of the step cycle.

The electrical stimulation was applied to the CP nerve and/or the tibial nerve by surface Ag-AgCl electrodes (Kendall LTP). The stimulus electrode for the CP nerve was placed at a low-threshold point near the neck of the fibula where activation of TA was prominent, and activation of peroneal muscles was weak, and the anode electrode was located below the patella. The electrodes for the tibial nerve stimulation were placed in the popliteal fossa and over the patella. Stimulus pulses were 1-ms rectangular pulses and delivered from Grass SD9 isolated stimulators (Grass Instruments, Quincy MA). The test stimulation was triggered by a random pulse generator so that the stimuli were generated with pseudorandom intervals (1.6–2.7 s).

Data analysis. To evaluate the effect of antagonist muscle nerve stimulation on the target muscle activity, the target muscle EMG was rectified, and the mean level of EMG amplitude was measured for a 7- to 10-ms period including the peak depression (Fig. 1). When the period of inhibition appeared to be longer than 20 ms, the first 10 ms were used for analyses. Because it is measured over a period of time, the inhibition may contain disynaptic and other short-latency reflex components. We will refer to this as short-latency reciprocal inhibition or simply reciprocal inhibition, without specifying neural pathways that may be involved. To measure the background EMG activity level for standing, a 40-ms prestimulus period was used. For walking and running, the average EMG activity was computed for unstimulated steps (>50 steps) and used as the control EMG (10, 49, 61). The amount of reciprocal inhibition was expressed as the difference in the mean rectified EMG level in the chosen 7- to 10-ms period from the background studied in 40 ms before stimulation or in the control EMG of unstimulated steps. On calculation of the amount of inhibition, some of the collected responses evoked by too strong or too weak stimulation (indicated by amplitudes of M waves) were eliminated from the further analysis.

To observe the extent of inhibition at different times in the step cycle, inhibition during walking or running was sorted into different
Fig. 1. Stimulating an antagonist muscle nerve decreases ongoing voluntary EMG activity. Reciprocal inhibition of the soleus EMG activity was observed after the H reflex in the tibialis anterior (TA; A) by stimulation at 1.5 × motor threshold (MT) of the common peroneal (CP) nerve. Note that the positive signal of 0–20 ms in the rectified EMG is pickup from activation of the CP nerve innervated muscles (e.g., extensor digitorum longus and peroneus longus). The mean level of inhibition (mV) was measured for a 7- to 10-ms window (see short horizontal bar) including the peak depression in the rectified EMG. (The amount of inhibition is indicated as a double-headed arrow.) The significance of inhibition was assessed by either the ratio or difference between the mean inhibition level and the mean rectified background EMG level. Background EMG levels were calculated from 40 ms of prestimulus period for standing data and from the corresponding 7- to 10-ms window of unstimulated steps for walking data. The long horizontal line indicates the background EMG level. Time 0 refers to the time of nerve stimulation. Twenty sweeps were averaged for each part of the figure.

bins according to the time at which the stimulation was given. For each subject, the step cycle was divided into 16 equal bins, and the inhibitory responses produced by stimuli within the same bin were averaged together (12, 13, 25).

RESULTS

Effects of stimulus intensity on inhibition of ongoing EMG activity. When the CP nerve stimulus intensity increased (Fig. 2A), the amount of inhibition in the soleus EMG increased (for calculation of inhibition, see MATERIALS AND METHODS). Although the TA H reflex could be evoked in some subjects as in Fig. 1, the amplitude was often small. Even in subjects without clear H reflexes, inhibition of the ongoing soleus EMG was observed. Thus the presence or absence of the H reflex seemed not to affect the amount of inhibition in the soleus EMG. Inhibition of the soleus EMG as a fraction of background EMG in all subjects is shown against the stimulus intensity expressed as a ratio to the TA MT in Fig. 2A. An exponential curve fitted by least mean squares method had a correlation coefficient of 0.60 and indicated the threshold for inhibition of 0.80. Thus the fibers responsible for the inhibition have a threshold lower than the M-wave threshold and are presumably primary muscle spindle afferents (1, 10, 37, 49). However, near the threshold, some subjects showed no inhibition, whereas some others showed some inhibition. Clear inhibition was observed in all subjects with stimulus intensities of ≥1.4 × MT.

The effect of tibial nerve stimulus intensity on the amount of inhibition in the TA was investigated in all 14 subjects. Inhibition of the TA normalized by the background EMG is plotted in relation to the tibial nerve stimulus intensity in Fig. 2B. A fitted exponential curve (r = 0.49) indicated the threshold for inhibition of 0.79, which is again below the M-wave threshold. Compared with the soleus, the stimulation-inhibition relation was less clear in the TA, although most subjects showed inhibition with the stimulation above 1.4 × MT. Thus a stimulus intensity of 1.5 × MT was mainly used in this study for both the soleus and TA, because it reliably produced inhibition of ongoing EMG activity.

In Fig. 3, reciprocal inhibition in the soleus EMG during standing and walking at 4 km/h is plotted against different background EMG levels at 1.5 (A) and 1.1 (B) × MT of stimulation. As shown in Fig. 3A, the higher the soleus background level, the larger the amount of inhibition with a stimulus intensity of 1.5 × MT. A dashed line is drawn that corresponds to 100% inhibition. The means ± SD of the correlation coefficients (Pearson’s r) for all subjects were 0.90 ± 0.08 for standing and 0.76 ± 0.17 for walking. Group means of regression slopes were significantly >0 for both standing (0.54 ± 0.13, P < 0.0001; Student’s 1-sample t-test) and walking (0.34 ± 0.12, P < 0.0001), indicating significant effects of background EMG level on the amount of inhibition and a significant difference between the tasks (P < 0.01; Student’s paired t-test). For a fair comparison between the tasks, regression lines were also calculated for the same background EMG range (from 13.1 ± 1.8 to 70.0 ± 5.8% MVC). The group mean ± SD of slopes for standing was 0.49 ± 0.15,
Fig. 3. Inhibition of ongoing EMG activity increases as the background EMG activity increases. The amount of inhibition as the difference between the mean rectified EMG of a selected 7- to 10-ms window and the background EMG is presented as a function of background EMG levels for standing and walking. To show the level of muscle activation, both the inhibition and background EMG are expressed as percent maximal voluntary tonic contraction (%MVC). Stimulus intensities of $1.5 \times MT$ (A) and $1.1 \times MT$ (B) were used to examine the effect of stimulus intensity on inhibition of ongoing EMG activity. Dashed lines are unity lines indicating complete suppression of ongoing EMG activity. Each point is the average of 4 responses that are the closest in the background EMG for standing or in the step cycle for walking. Values are slopes of regression lines (standing: solid, walking: dotted). Both A and B are from subject AK.

and it was significantly larger ($P < 0.05$; Student’s paired $t$-test) than that for walking (0.38 ± 0.12).

The reciprocal inhibition was also examined in seven subjects by using a stimulus intensity of $1.1 \times MT$. Figure 3B is an example of the soleus inhibition induced by $1.1 \times MT$ of the CP nerve stimulation in a single subject. In this subject, the amount of inhibition increased with an increase of the background EMG level in both standing and walking; slopes of regression lines were 0.35 for standing and 0.28 for walking (not significantly different). The group means ± SD were 0.36 ± 0.12 for standing and 0.26 ± 0.12 for walking, and there was no significant difference between the tasks ($P = 0.06$). When slopes of regression were compared for the same range of background EMG activity (from 12.3 ± 1.1 to 69.4 ± 6.3% MVC), the means ± SD were 0.30 ± 0.09 for standing and 0.25 ± 0.07 for walking, again showing no significant difference ($P = 0.10$). The amount of inhibition indicated by the slope was larger with $1.5 \times MT$ than with $1.1 \times MT$ in standing ($P < 0.001$) but not in walking ($P = 0.30$).

Similarly, for the soleus, the amount of reciprocal inhibition in the TA was examined with different background EMG levels at stimulus intensities of $1.5$ and $1.1 \times MT$. The correlation coefficients were 0.73 ± 0.22 for standing and 0.63 ± 0.35 for walking at 4 km/h. Slopes of regression lines were significantly $>0$ for both standing ($0.45 \pm 0.24$, $P < 0.0001$) and walking ($0.43 \pm 0.28$, $P < 0.0001$), and they were not different between the tasks ($P = 0.68$). When regression lines were calculated for the same background EMG range (from 8.2 ± 0.9 to 33.4 ± 2.9% MVC) for a fairer comparison, the group means ± SD of slopes were 0.40 ± 0.24 for standing and 0.44 ± 0.28 for walking, and not different between the two tasks ($P = 0.41$). Inhibition of the TA EMG was also examined with a stimulus intensity of $1.1 \times MT$. When regression lines were compared between the tasks, slopes were not significantly different (standing: $0.39 \pm 0.18$, walking: $0.24 \pm 0.10$, $P = 0.10$). When the slope comparison was made for the same background EMG range (from 6.5 ± 2.0 to 29.3 ± 4.7% MVC), the difference was less (standing: $0.34 \pm 0.22$, walking: $0.24 \pm 0.09$, $P = 0.37$). Thus the task-dependent modulation of reciprocal inhibition was not demonstrated with stimulation of the tibial nerve at either $1.5$ or $1.1 \times MT$. Different from the observation for inhibition of the soleus EMG, the slope of inhibition was not significantly different between $1.5$ and $1.1 \times MT$ in either standing ($P = 0.09$) or walking ($P = 0.08$).

Overall, the same trends were seen with $1.1 \times MT$ as for $1.5 \times MT$, but the amount of inhibition was larger for $1.5 \times MT$ and the differences between conditions were statistically significant. Therefore, we used $1.5 \times MT$ to investigate task-dependent modulation of reciprocal inhibition.

Inhibition of ongoing EMG activity during walking and running. Inhibition of the ongoing EMG activity was investigated in eight subjects by stimulating either the CP nerve or the tibial nerve by using a stimulus intensity of $1.5 \times MT$ during standing, walking at either 3 or 6 km/h, and jogging or running at either 6 or 9 km/h. The means ± SD of the step cycle duration were 1,261 ± 112 ms for slow walking (3 km/h), 984 ± 48 ms for fast walking (6 km/h), 801 ± 42 ms for slow running (6 km/h), and 762 ± 33 ms for running (9 km/h).

The time course of change in the soleus inhibition for each locomotor condition is shown in Fig. 4, A, C, E, and G. The step cycle was divided into 16 equal bins, and inhibitory responses produced by stimuli within the same bin were averaged together for each subject. Approximately 10 responses were contained in each bin. The mean rectified EMG (i.e., background EMG level) calculated in unstimulated steps and the anticipated amount of inhibition were also averaged for each bin, and they were plotted with the actual measure of inhibition. The anticipated inhibition was calculated from the standing condition for a given level of background EMG activity and was reasonably reliable, as the correlation coefficient of linear regression between the background EMG level, and the amount of inhibition was high for standing ($r = 0.88 \pm 0.04$, mean ± SE).

As can be seen in Fig. 4, A, C, E, and G, the actual amount of inhibition never exceeded the anticipated inhibition, although the difference between the anticipated and actual inhibition changed, depending on the background EMG level and locomotor condition. The difference was small for slow walking, whereas it was large for running. Results of one-way repeated-measures ANOVA performed on the inhibition in 16 bins revealed significant effects of time course in the step cycle on the amount of inhibition for all four locomotor conditions ($P < 0.0001$). However, when the inhibition was expressed as a fraction of the background activity level, no single bins were significantly different from others.
Similarly, the time course of the TA inhibition is presented in Fig. 4, B, D, F, and H, together with the amount of inhibition estimated from standing condition and the background EMG activity. A high correlation coefficient of linear regression between the background activity and the amount of inhibition ($r = 0.96 \pm 0.01$) for standing likely gave reliable estimates of inhibition to a given background EMG level. The amount of inhibition actually measured was always smaller than the anticipated amount, indicating the difference in reciprocal inhibition between standing and walking or running. Significant effects of time course on the inhibition were detected by one-way repeated-measures ANOVA performed on 16 bins for all 4 conditions ($P < 0.0001$). When the amount of inhibition was normalized by the background activity, however, there was no difference among bins.

Inhibition of the ongoing soleus EMG activity was expressed as a function of background EMG activity for five different conditions. Group means of regression slopes, calculated for the full range of background EMG activity, are presented in Fig. 5A. With increasing speed (from standing, walking, to running), the slope becomes less steep and the correlation becomes weaker. The result of one-way repeated-measures ANOVA showed a significant effect of condition on the slope ($F = 16.6, P < 0.0001$). Slopes were also compared between two conditions using Student’s paired t-test. Significant differences between the conditions are indicated in Fig. 5A (by *$P < 0.05$ or **$P < 0.01$). Note that some of the differences might appear to be significant by chance because 10 paired comparisons were made between tasks. However, most conditions were highly significantly different. For example, the slope for standing (mean $\pm$ SE: $0.515 \pm 0.050$) was steeper than those for walking at 6 km/h ($0.305 \pm 0.043$), running at 6 km/h ($0.199 \pm 0.040$), and running at 9 km/h ($0.269 \pm 0.040$) but not for walking at 3 km/h ($0.411 \pm 0.062$).
Fig. 5. Amount of inhibition decreases from standing, to walking, to running. A: group means of regression slopes between the background EMG and the amount of soleus inhibition are presented with SE bars. With increasing speed, the slope becomes less and correlation becomes weaker. B: group means of slopes for the TA inhibition are presented with SE bars. A stimulus intensity of 1.5 × MT was used for all conditions. Significant difference between 2 conditions tested by Student’s paired t-test: *P < 0.05 or **P < 0.01.

No significant difference was seen between running at 6 and 9 km/h. The mean ± SE of the correlation coefficients between the background EMG and the amount of inhibition was 0.87 ± 0.04 for standing, 0.75 ± 0.07 for walking at 3 km/h, 0.75 ± 0.05 for walking at 6 km/h, 0.55 ± 0.09 for running at 6 km/h, and 0.52 ± 0.08 for running at 9 km/h. Although the correlation became less when condition changed from standing, to walking, to running, the correlations were all statistically significant.

The correlation between inhibition of the ongoing TA EMG activity and the background EMG activity was evaluated for each condition. Group means of regression slopes are presented in Fig. 5B. The result of ANOVA revealed a significant effect of condition on the steepness of regression slope (F = 16.1, P < 0.0001). The mean ± SE of individual slopes for standing was 0.678 ± 0.047, and it was significantly steeper than those for walking at 3 km/h (0.492 ± 0.047) and 6 km/h (0.423 ± 0.049) and running at 6 km/h (0.360 ± 0.064) and 9 km/h (0.354 ± 0.068). The difference between walking at 3 km/h and running at 6 km/h (P < 0.05) was also significant.

The correlation coefficient between the background EMG and the amount of inhibition decreased from standing, to walking, to running: mean ± SE, 0.96 ± 0.01 for standing, 0.73 ± 0.08 for walking at 3 km/h, 0.75 ± 0.05 for walking at 6 km/h, 0.67 ± 0.07 for running at 6 km/h, and 0.63 ± 0.07 for running at 9 km/h. All of these values were statistically significant.

**DISCUSSION**

The main goal of this study was to examine spinal inhibition induced by the antagonist muscle nerve stimulation in relation to a task dependence. The effect of stimulus intensity on the amount of inhibition was examined to choose a proper stimulation to study the task-dependent modulation of inhibition.

**Effect of stimulus intensity on the amount of inhibition.** The depression in the rectified EMG increased significantly with the level of activity in both the soleus and TA muscles for our group of subjects with stimulation of the CP and tibial nerves, respectively (Fig. 3), in agreement with previous work (10, 49). The slope relating the inhibition and the background EMG activity with CP stimulation at 1.5 × MT was significantly greater during standing than walking at 4 km/h (54% compared with 34%, 11 subjects) but not at 1.1 × MT (36% compared with 26% in 7 subjects). These results are opposite to those of Petersen et al. (49), who found a significant difference at 1.1 × MT (14 subjects) but not at 1.6 × MT (4 subjects). The reason for the discrepancy between the studies is not known. We concentrated on the higher stimulus level because the amount of inhibition increased with stimulus intensity and could then be observed in all subjects (Fig. 2). With stimuli close to MT little inhibition was observed in some subjects, so the ability to see differences without averaging very large numbers of stimuli was severely compromised. When the tibial nerve was stimulated, no significant difference in inhibition was observed in the TA muscle between standing and walking with either 1.1 or 1.5 × MT. Petersen et al. (49) did not report any data on this type of stimulation. A stimulus intensity of 1.5 × MT was thus used to study reciprocal inhibition in several different types of movement.

Recently, the axonal excitability of motoneurons has been shown to be activity dependent (8, 33–35, 59). Also, it has been reported that the maximum M-wave changes during walking and running (53, 55) or during a long-lasting experiment (16). Thus there was a possibility that constant stimuli that produce the same amplitude of M waves might not be activating the same population of neurons between and within dynamic motor tasks. However, recently Ferris et al. (27) showed that the maximum M-wave amplitude changed only up to 20–30% during walking and running. Also, as shown in Fig. 2A, the amount of inhibition reached a plateau with stimulus intensities ≥1.4 × MT. Finally, maintaining the size of M wave was the best we could do to keep the stimulus intensity constant. We are confident that enough afferents were recruited to activate inhibitory interneurons throughout an experiment.

**EMG activity and correlation coefficient.** The most important finding in this study was that the amount of inhibition, indicated by a slope of linear regression between the background EMG and the amount of inhibition, decreased from standing, to walking, to running (Fig. 5). However, whereas the slope of regression changed from standing to walking to running, the correlation coefficient also became smaller from standing to locomotion. Perhaps, the reduced slope of inhibition could be due to increased variation in the EMG activity. In fact, the EMG activity level changes more rapidly with increasing speed of locomotion, and that will likely increase the variability of EMG activity and reduce the correlation coefficient. However, Pearson’s r remained in a statistically signifi-
significant range over different conditions. Therefore, the calculated slopes of regression lines were still reliable, and a change in the regression slope among different conditions cannot be explained fully by the variability of EMG.

In the present study, the range of background EMG level varied from one condition to another. For instance, the soleus EMG range during running at 9 km/h was three times larger than the range during walking at 3 km/h. So one might ask whether the difference in the range of background EMG activity among different conditions influenced the observed task-dependent modulation of inhibition. However, a slope change (i.e., a change in the amount of inhibition) seemed less likely to be a simple function of background EMG level, because a similar task-dependent modulation was observed in both the TA inhibition, in which the background EMG range was standing > running > walking, and the soleus inhibition, in which the EMG range was standing = walking < running.

Spinal inhibition and phase of the step cycle. Previous work has found a task dependence for inhibiting the soleus H reflex in the swing phase of walking, compared with standing, by measuring the effect of voluntary TA activity (36) or of CP nerve stimulation (49) in a few subjects (4 of 30) who showed an H reflex during the swing phase of walking. We have not repeated those experiments but compared the amount of inhibition in equally separated 16 bins for 4 different locomotor conditions. Although significant effects of time course in the step cycle were found for all four locomotor conditions with the CP nerve stimulation, when the inhibition was normalized by the background activity, no single bins showed significantly more or less inhibition than other bins in any conditions (Fig. 4). Inhibition of the TA was also compared among 16 bins separated according to the time course of step cycle. Because the early to midswing phase (i.e., first burst) and the late swing to transition phase (i.e., second burst) of the TA activity have functionally different roles in human walking and are differentially modulated in response to cutaneous inputs (18, 22, 23, 29, 61), inhibition of these two bursts might be modulated differently. However, the relative amount of inhibition to the background activity was not significantly different over the step cycle. Thus the inhibition did not depend on the part of the stance phase for the soleus or the part of the swing phase for the TA. In other words, these results assured us that the data for the whole step cycle could be pooled, because the inhibition depended mostly on the background EMG level and not on the phase in the step cycle.

**Task dependence of reciprocal inhibition.** In this study, we compared the task dependence of spinal reciprocal inhibition at a short latency with different stimulus intensities, stimulation of different nerves, different analytic procedures, and a range of tasks from standing to walking to running. The results are summarized in Table 1. Although there are some values that are significant at the $P < 0.05$ level or the $P < 0.01$ level, many are not significant. These observations differ from the task dependence in the H reflex (12, 13, 25) in which the reflex during standing could be up to 3.5 times larger than during walking and the reflex gain was substantially decreased from walking to running at the same speed (6 km/h). However, some of these results have not been reproduced in a recent study (27). Similarly, dramatic differences are seen in other reflexes. For example, Duysens et al. (21) compared cutaneous reflex during standing and running and found a directional difference between the tasks; the response in the TA was predominantly suppressive during standing and facilitatory during running. Similarly, Pearson et al. (45–47) found that the inhibition, arising largely from Golgi tendon organs during standing, could be reversed to an excitation during some phases of walking. In the present study, inhibition was never reversed to facilitation across different tasks. We conclude that there is some evidence for a task dependence of reciprocal inhibition but that it is weaker than for other spinal reflexes.

The rationale for a task dependence is clear for these other reflexes. For example, the calf muscles are stretched under the weight of the body during stance, and the stretch reflex is useful in pushing the body off the ground at the end of stance. However, the muscles are also stretched during the swing phase, so, without reflex modulation, a reflex would extend the ankle and perhaps cause the foot to hit the ground (12, 51, 62). Similarly, a cutaneous input during swing phase leads to a flexion reflex that can prevent a stumble, but when the limb is striking the ground at the end of stance, a flexion could cause a fall. An extension is needed to support the body (61, 63–65). Finally, as long as a limb is loaded during walking, extensors must remain active to support the body weight, and positive feedback from Golgi tendon organs would be very useful for this purpose (19, 20, 30, 44, 45, 60).

**Effects of speed and functional role.** We cannot envisage a similar functional role for task dependence of a spinal reciprocal inhibition. Although the task dependence was weak, when we examined the full range of speeds in Fig. 5, there was a large and consistent decrease in the inhibition with speed.

**Table 1. Task dependence of reciprocal inhibition**

<table>
<thead>
<tr>
<th>Nerve Stimulation</th>
<th>Task</th>
<th>Difference</th>
<th>Data Compared</th>
</tr>
</thead>
<tbody>
<tr>
<td>CP (1.5 × MT)</td>
<td>Standing vs. walking (4 km/h)</td>
<td>$P &lt; 0.01$</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>CP (1.5 × MT)</td>
<td>Standing vs. walking (4 km/h)</td>
<td>$P &lt; 0.05$</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>CP (1.1 × MT)</td>
<td>Standing vs. walking (4 km/h)</td>
<td>NS</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>CP (1.1 × MT)</td>
<td>Standing vs. walking (4 km/h)</td>
<td>NS</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>Tibial (1.5 × MT)</td>
<td>Standing vs. walking (4 km/h)</td>
<td>NS</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>Tibial (1.5 × MT)</td>
<td>Standing vs. walking (4 km/h)</td>
<td>NS</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>Tibial (1.1 × MT)</td>
<td>Standing vs. walking (4 km/h)</td>
<td>NS</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>CP (1.5 × MT)</td>
<td>Standing vs. walking (3 km/h)</td>
<td>NS</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>CP (1.5 × MT)</td>
<td>Walking (6 km/h) vs. running (6 km/h)</td>
<td>$P &lt; 0.01$</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>CP (1.5 × MT)</td>
<td>Walking (6 km/h) vs. running (6 km/h)</td>
<td>$P &lt; 0.05$</td>
<td>All values Matched EMG levels</td>
</tr>
<tr>
<td>Tibial (1.5 × MT)</td>
<td>Walking (6 km/h) vs. running (6 km/h)</td>
<td>NS</td>
<td>All values Matched EMG levels</td>
</tr>
</tbody>
</table>

CP, common peroneal; EMG, electromyograph; NS, not significant.
This makes sense because, as the speed is increased, there will be less time in a given phase for the spinal inhibition to act. In addition, for rapid movements many muscles need to be activated maximally. As a muscle is rapidly shortening, muscle spindles in the antagonist muscles will be powerfully stretched, activated maximally. As a muscle is rapidly shortening, muscle addition, for rapid movements many muscles need to be less time in a given phase for the spinal inhibition to act. In this work was supported by the Canadian Institutes of Health Research and the Alberta Heritage Foundation for Medical Research.

ACKNOWLEDGMENTS

We thank Dr. Charles Capaday, Dr. Ming Chan, Dr. Monica Gorassini, and Dr. Jaynie Yang for helpful comments on a draft of the manuscript.

GRANTS

This work was supported by the Canadian Institutes of Health Research and the Alberta Heritage Foundation for Medical Research.

REFERENCES


