Differential Control of Reciprocal Inhibition During Walking Versus Postural and Voluntary Motor Tasks in Humans

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Lavoie, Brigitte A., Hervé Devanne, and Charles Capaday. Differential control of reciprocal inhibition during walking versus postural and voluntary motor tasks in humans. *J. Neurophysiol.* 78: 429–438, 1997. Experiments were done to determine whether the strength of reciprocal inhibition from ankle flexors to extensors can be controlled independently of the level of ongoing motor activity in a task-dependent manner. In this paper we use the term reciprocal inhibition in the functional sense—inhibition of the antagonist(s) during activity of the agonist(s)—without reference to specific neural pathways that may be involved. The strength of reciprocal inhibition of the soleus α-motoneurons was determined by measuring the amplitude of the H reflex during voluntary, postural, and locomotor tasks requiring activity of the ankle flexor tibialis anterior (TA). Differences in the strength of reciprocal inhibition between tasks were determined from plots of the soleus H reflex amplitude versus the mean value of the TA electromyogram (EMG). Additionally, in tasks involving movement, the correlation between the H reflex amplitude and the joint kinematics was calculated. In most subjects (15 of 22) the soleus H reflex decreased approximately linearly with increasing tonic voluntary contractions of the TA. The H reflex also decreased approximately linearly with the TA EMG activity when subjects where asked to lean backward. There were no statistical differences between the regression lines obtained in these tasks. In some subjects (7 of 22), however, the H reflex amplitude was independent of the level of TA EMG activity, except for a sudden drop at high levels of TA activity (~60–80% of maximum voluntary contraction). The type of relation between the soleus H reflex and the TA EMG activity in these tasks was not correlated with the maximum H reflex to maximum M wave ($H_{\max}/M_{\max}$) ratio measured during quiet standing. In marked contrast, during the swing phase of walking—over the same range of TA EMG activity as during the tonic voluntary contraction task—the H reflex was reduced to zero in most subjects (24 of 31). In seven subjects the H reflex during the swing phase was reduced to some 5% of the value during quiet standing. The same result was found when subjects were asked to produce a stepping movement with one leg (OLS) in response to an auditory "go" signal. Additionally, in the OLS task it was possible to examine the behavior of the H reflex during the reaction time and thus to evaluate the relative contribution of central commands versus movement-related afferent activity to the inhibition of the soleus H reflex. In 11 of 12 subjects the H reflex attained its minimum value before either the onset of EMG activity or movement of any of the leg joints. It is significant that the H reflex was most powerfully inhibited during the swing phase of walking and the closely related OLS task. The H reflex was also measured during isolated ankle dorsiflexion movements. The subjects were asked to track a target displayed on a computer screen with dorsiflexion movements of the ankle. The trajectory of the target was the same as that of the ankle during the swing phase of walking. The soleus H reflexes were intermediate in size between the values obtained in the tonic contraction task and the walking or OLS tasks. A negative, but weak, correlation ($r^2 < 0.68$) between the soleus H reflex and the TA EMG was found in 3 of 10 subjects. Furthermore, there was no correlation between the H reflex amplitude and the ankle angular displacement or angular velocity. In this task, as in the OLS task, the H reflex began to decrease during the reaction time before the onset of TA EMG activity. We conclude that the strength of reciprocal inhibition of the soleus α-motoneuron pool can thus be controlled independently of the level of motor activity in the ankle flexors. The strength of the inhibition of the antagonist(s) depends on the task, and for each task the strength of the inhibition is not necessarily proportional to the level of motor activity in the agonist(s). Additionally, the evidence suggests a strong central contribution to these task-dependent changes, because the inhibition of the H reflex is essentially completed during the reaction time before the onset of EMG activity or joint movement. The possible neural mechanisms involved in the task-dependent control of reciprocal inhibition are treated in the DISCUSSION.

INTRODUCTION

Reciprocal inhibition between antagonist muscles is a fundamental aspect of the neural basis of motor control. Disruptions of reciprocal inhibition are an important feature of human spasticity, a condition in which involuntary cocontractions of antagonist muscles, rather than selective activation of agonists, often occur (Ashby and Wiens 1989: Boorman et al. 1991). It is important to distinguish the phenomenon of reciprocal inhibition—relaxation of the antagonist muscle during activity of the agonist (Sherrington 1913)—from the neural mechanisms that produce it. Reciprocal inhibition is mediated, at least in part, by a disynaptic circuit in the spinal cord that is subject to several supraspinal as well as segmental modulatory mechanisms (reviewed by Jankowska 1992). Several other neural mechanisms, such as presynaptic inhibition, have been suggested to be involved in reciprocal inhibition (Crone and Nielsen 1989; El Tohamy and Sedgwick 1983; Fu et al. 1978). In this paper we use the term reciprocal inhibition in the functional, or descriptive, sense defined above. We use the term disynaptic inhibitory pathway when making specific reference to this spinal circuit.

Regardless of the ensemble of neural mechanisms that may be involved, the need for controlling the strength of reciprocal inhibition becomes apparent when considering the variety of ways in which antagonist muscles are activated. Synergies between antagonist muscles include simple patterns of reciprocal activation, cocontractions, and complex triphasic activation patterns. Indeed, it was recently shown that the strength of the disynaptic reciprocal inhibition is reduced during cocontractions of antagonist muscles compared with reciprocal activation (Nielsen and Kagamihara

1992; see Nielsen and Kagamihara 1993 and Nielsen et al. 1994 for studies of presynaptic inhibition and stretch reflexes). Nonetheless, nearly all of what is known about how the reciprocal inhibitory pathway(s) is controlled comes from experiments performed at the wrist or ankle during voluntary isometric contractions in which agonists and antagonists are activated in a reciprocal manner (see review by Crone and Nielsen 1994). In general, the body of experimental results is consistent with the hypothesis of Lundberg (1970), specifically formulated for the reciprocal disynaptic pathway, which asserts that ". . . voluntary activation of a muscle(s) is linked to a proportional inhibition of its antagonist(s)." In addition, it has also been reported that during tonic voluntary contractions the Ia-inhibitory interneurons (IaIns) projecting to the antagonist motoneurons appear to be more excitable than at rest (Crone and Nielsen 1994; Nielsen et al. 1995; Sinkjaer et al. 1995). This observation supports the idea that during voluntary motor activity supraspinal and afferent inputs converge on these interneurons as suggested by Lundberg (1970).

In contrast to most studies of this problem, in which the strength of reciprocal inhibition acting on the antagonist motoneurons is measured, we investigated whether the reciprocal inhibitory pathway to an active motoneuron pool remains operational (Capaday et al. 1990). It was shown that the reciprocal disynaptic pathway from the ankle flexors to the soleus remained operational during tonic voluntary activity of the soleus, as well as during the stance phase of locomotion. There was no difference in the strength of the inhibition between these two tasks, i.e., when the target muscle was active. It was thus suggested that the reciprocal inhibitory pathway to an active motoneuron pool remains operational so as to contribute to the rapid and timely cessation of motor activity, such as occurs at the transition from stance to swing, on the basis of the exact kinematic events during task performance (Capaday et al. 1990).

The issue addressed in this study is how reciprocal inhibition is modulated during motor tasks when the target muscle is inactive, that is, when it acts as a "silent" antagonist. Specifically, we asked whether the strength of reciprocal inhibition from ankle flexors to extensors can be controlled independently of the level of ongoing motor activity in a task-dependent manner. To this end, the strength of reciprocal inhibition was determined in a variety of motor tasks including locomotor, postural, and voluntary activities (Fig. 1). The soleus H reflex was measured at different levels of activity of the tibialis anterior (TA) in each of these tasks and also correlated with the joint kinematics. We found that the strength of the reciprocal inhibition of the soleus H reflex was very markedly dependent on the motor task, and we present evidence that this control is principally determined by central neural mechanisms. A short account of these findings has been published as an abstract (Lavoie et al. 1995).

METHODS

This study was performed on 23 healthy human subjects ranging in age between 21 and 34 yr. Each subject was studied in at least two motor tasks, and in up to four tasks, during a single session. Nine subjects came back for a second session during which they were studied in two other tasks. The exact number of subjects studied in each task is given in the appropriate part of the RESULTS section. All subjects gave their consent after being informed of the nature and procedures of the experiments. The study was conducted in accordance with the declaration of Helsinki and approved by the local ethics committee.

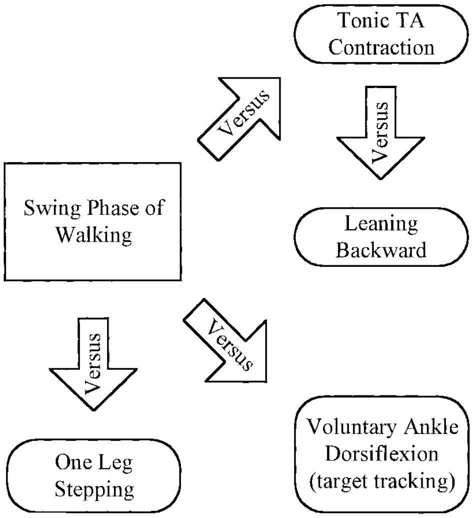

FIG. 1. Schematic summary of tasks in which soleus H reflex was studied during activity of its antagonist the ankle flexor tibialis anterior (TA). H reflex was measured during swing phase of walking and served as a basis of comparison with either voluntary movement tasks, tonic TA contractions, or postural maintenance requiring TA activity. In tasks involving movement of the ankle, or of the whole leg, subjects were required to track the ankle trajectory produced during the swing phase of their own walking cycle (i.e., target tracking).

Electrical stimulation

The tibial nerve was electrically stimulated with square pulses 0.5 ms in duration delivered through a constant voltage stimulus isolation unit. The cathode (Ag-AgCl electrode 0.7 mm diam) was placed on the skin overlying the nerve at an optimal spot for eliciting an H reflex in the soleus. The anode, a large metal plate (3 × 7 cm) covered in gauze and moistened with saline, was placed on the opposite side of the leg above the patella. The cathode was covered by an elastic rubber strap wrapped around the leg and tightened so as to maintain pressure on it. The stimulus intensity used to elicit H reflexes was in the plateau range of the H reflex recruitment curve, at a point at which a small M wave can be obtained (Capaday and Stein 1986). In our sample of subjects the H reflex plateau was on average 45 ± 16% (mean ± SD) of maximum M wave (M_{max}).

Electromyographic recordings

The electromyographic (EMG) activity of the soleus and TA muscles were recorded with bipolar surface Ag-AgCl electrodes, each 0.7 mm diam. In some cases the EMG activity of the knee extensor vastus lateralis was also recorded. The electrodes were positioned 2–3 cm apart on the respective muscle bellies. The ground electrode, a large metal plate (3 × 9 cm) covered in gauze and moistened with saline, was placed over the muscle bellies of the gastrocnemii between the stimulating and the recording electrodes and connected to the common input of optically isolated preamplifiers. The M wave and H reflex responses of the soleus were amplified, high-pass filtered at 20 Hz, and low-pass filtered at 1 kHz, with single time constant filters. The level of activity of the soleus and TA was estimated from the mean value of the surface EMG signal, high-pass filtered at 20 Hz, rectified, and low-pass filtered at 100 Hz. The ankle, knee, and hip angular displacements were measured with goniometers. All signals were digitized in real time at a sample rate of 5,000 points per second.

The data acquisition program had a time-amplitude window discriminator subroutine that automatically rejected trials in which the tibial nerve stimulus did not produce an M wave that fell within specified limits (Capaday et al. 1995). With this method the coefficient of variation of the averaged M waves is typically between 0.1 and 0.15 (i.e., ~0.5–1% of $M_{\max}$). Thus for each subject H reflexes were evoked by stimuli of very nearly the same intensity in all tasks studied.

Additional details and a discussion of the biophysical and physiological principles of the methods for eliciting and analyzing reflex responses in freely moving subjects can be found in a recent review article (Capaday 1997).

Motor tasks

During an experimental session, each subject performed at least two of the six motor tasks described below. Three subjects were studied in four tasks in a single experimental session.

SWING PHASE OF WALKING. All subjects were asked to walk on a treadmill at their own preferred speed (4–5 km/h) at the beginning of an experimental session. Once a stable walking cadence was established, the step cycle EMG patterns of the soleus and TA and the joint displacements were acquired and averaged ($n = 100$) in real time. The onset of the swing phase relative to heel contact and the mean value of the TA EMG during swing were determined from these measurements. The angular displacement of the ankle produced during swing phase was extracted from the digitized records and processed by the software to give the subjects a visual display of the ankle trajectory they were required to follow during the one-leg stepping (OLS) and voluntary ankle dorsiflexion tasks. In other words, the subjects were required to reproduce the ankle movements that occurred during the swing phase of walking. To determine the amplitude of the soleus H reflex during the swing phase of walking (when the ankle extensors are inactive) stimuli were delivered to the tibial nerve at five to seven phases during swing. Typically, four to eight reflex responses were averaged at each phase of swing.

OLS WITH ANKLE MOVEMENT. OLS was chosen as a voluntary motor task that strongly resembles the swing phase of walking, e.g., activation of the TA to clear the foot off the ground. Additionally, changes in the H reflex amplitude can be studied during the reaction time as well as at various times during the movement. Fourteen subjects were asked to perform the OLS task while tracking the ankle trajectory of the swing phase of their own walking cycle displayed on a computer monitor. The subject was supported by the contralateral (left) leg, as during the swing phase of walking. At the starting position, the right leg was behind the subject in nearly the same configuration (i.e., joint angles) as at the transition between the stance and swing phases of walking. The ankle extensors were inactive at the initial position (Fig. 6). The subjects were instructed to swing the leg after hearing a ''go'' signal (1 kHz, 1 s in duration) and to put the heel down in front of themselves at the same ankle angle as at heel contact during walking, and then to return to the initial position. The auditory go signals were given randomly every 3–7 s. Trials that were within ±5° of the control ankle trajectory produced a second auditory tone to indicate to the subject that the trial was accepted by the computer. This interactive method allowed the subjects to learn within a few minutes to execute the task within the prescribed limits. Additionally, to further help the subjects, markers were placed on the floor to indicate the starting position behind and the final position in front of themselves. These markers considerably helped the subject in the OLS task. The tibial nerve was stimulated at random times after the auditory go signal—one stimulus per movement. The time course of the soleus H reflex inhibition was measured in 10-ms increments during the reaction time before TA EMG onset and in increments of 50 ms after the onset of TA activity, until the end of movement. Typically, four to eight reflex responses were averaged at each time interval.

OLS WITHOUT ANKLE MOVEMENT. This task was chosen to determine the role of the ankle dorsiflexion per se on the modulation of the soleus H reflex during OLS and, by analogy, potentially during walking. Five subjects were instructed to execute the OLS task with the ankle fixed in an orthosis at a angle of 90°. It is notable that with the ankle fixed, thus eliminating the need to activate the TA, the subjects nonetheless automatically activated the TA even when instructed to relax. It was very difficult to silence the TA muscle during this task. In some cases, the subjects were asked to consciously attempt to activate the TA during the leg swing. This produced an enhanced activation of the TA over and above the activation that occurred automatically. Because the ankle was fixed, the leg motion was monitored by a goniometer at the knee. The floor markers guided the subjects to produce a leg swing of the required amplitude. The time course of the soleus H reflex was determined as described above. Typically, four to eight reflex responses were averaged at each time interval.

VOLUNTARY ANKLE DORSIFLEXION. Ten subjects were asked to reproduce the ankle movement of the swing phase of walking while seated on a high stool allowing the studied leg to be extended. The ankle was unrestrained and supported at the heel. The initial plantarflexed position of the ankle corresponded to the beginning of the swing phase of walking and the final ankle position was that at heel contact. The subjects were instructed to react to an auditory cue and to follow the trajectory displayed on a computer screen and then return to the initial position. Here again, the auditory cues were delivered randomly every 3–7 s. Each trial that was within ±5° of the control ankle trajectory triggered a second auditory tone to indicate to the subject that the trial was accepted. The time course of the soleus H reflex inhibition was determined starting at a time just before the onset of the TA EMG, which was measured from the average reaction time in trials without nerve stimulation.

VOLUNTARY TONIC TA CONTRACTION. The soleus H reflex was also measured at various levels of tonic TA voluntary activity in 17 subjects. The subjects stood with the tested foot placed under a rigid support that allowed the subjects to activate the TA nearly isometrically. In some cases the subjects were seated with the tested leg slightly extended (knee at ~120° and ankle at ~30°) and the foot placed under the rigid stop. Similar results were obtained with the two methods. The subjects were required to maintain different prescribed levels of TA EMG activity, which was displayed on an analog meter. The meter was calibrated so that a full-scale deflection of the needle corresponded to the maximum tonic contraction of the muscle, as measured by the rectified and filtered surface EMG (bandpass 10–20 Hz). The required contraction strengths varied from 10% of the maximum voluntary contraction (MVC) to ~80% of MVC. In this as in all other tasks studied, the soleus was silent. When the appropriate level was attained, soleus H reflexes were elicited at random intervals between 2 and 5 s. Typically, eight reflex responses were averaged at each contraction strength.

LEANING BACKWARD. Finally, the effect of postural changes requiring TA activity on the soleus H reflex was determined in four subjects. The subjects placed both feet under a support that allowed them to stabilize their posture during the trials. When subjects were asked to gradually lean backward, the activity of the TA increased over a range of levels comparable with that produced during voluntary tonic activity. The level of TA EMG activity was displayed on an analog meter placed in front of the subject. When the appropriate level of activity was attained, soleus H reflexes were elicited every 2–5 s. Typically, eight reflex responses were averaged at each contraction level.

Data reduction and analysis

The regression line and correlation coefficient relating the peak-to-peak amplitude of the soleus H reflex and the mean value of the TA rectified EMG were calculated for each task. In tasks involving movement, the linear regression parameters and the correlation coefficient between the H reflex amplitude and the joint kinematic variables were also calculated. The statistical significance of the correlation coefficients was determined. The deviation of the data points from a straight-line fit was evaluated by a runs test (Bendat and Piersol 1986). This statistical method determines the number of runs in the data points—a run is a consecutive series of residuals above or below the estimated best fitting line—and compares these with the expected number of runs on the basis of probabilistic rules. For each subject, the statistical difference between regression lines obtained in the different tasks was determined by an analysis of covariance (Kerlinger and Pedhazur 1973). Finally, in tasks involving a reaction time the amplitude of the H reflex was plotted against time after the go signal or relative to onset of the TA EMG. This was done to determine the timing of the H reflex inhibition relative to the EMG activities and leg kinematics.

Additionally, to determine the statistical association between ratio or ordinal variables and nominal variables, the nonparametric Mann-Witney *U* test and the Wald-Wolfowitz runs test were used.

Summary of the experimental protocols and data analysis

A schematic summary of the tasks investigated and of the comparisons made between the tasks is shown in Fig. 1. Briefly, the experiments involved measuring the soleus H reflex amplitude in a variety of motor tasks involving activity of its antagonist, the TA. For each task the amplitude of the H reflex was measured along with the level of EMG activity in the TA. In tasks involving movement, the joint kinematics were also recorded. In all cases, the H reflex was plotted against the mean value of the TA EMG measured over a 50-ms time segment just before the tibial nerve stimulus. In cases involving joint movement, the H reflex was also plotted against the angular displacement and velocity of the ankle, knee, or hip as appropriate to the task. Finally, in the voluntary movement tasks the time course of the H reflex inhibition was determined starting at the time of the auditory go signal. From these measurements the nature of the dependence of reciprocal inhibition of the H reflex was determined. These experiments involved very tight control of the required movement trajectory, the background level of TA EMG activity, and the M wave amplitude that served as a measure of stimulus intensity.

RESULTS

Two main observations on reciprocal inhibition of the soleus H reflex during activity of the TA were made in this study. In the first part of this section we show that the strength of the inhibition was very much dependent on the motor task. In the second part we present data addressing the issue of whether these task-dependent differences in the strength of reciprocal inhibition are the result of central commands, versus being subsequent to movement-related afferent activity (reafference).

Task dependence of the strength of reciprocal inhibition

In three subjects it was possible to investigate the four principal motor tasks in a single experimental session. Data from one of these subjects illustrating the main findings of this study are shown in Fig. 2. When the amplitude of the soleus H reflex is plotted against the mean value of the rectified TA EMG, the data points fall along a different curve for each task (Fig. 2). In most subjects (15 of 22), during voluntary tonic activity of the TA, the soleus H reflex decreases approximately linearly as a function of the TA EMG level (Figs. 2, 4, and 5). In a few subjects (7 of 22) these variables were not significantly correlated, except for a sudden drop at high levels of TA activity (~60–80% of MVC). The type of relation between the soleus H reflex amplitude and the TA EMG activity was independent of the maximum H reflex (H_{max})/M_{max} ratio measured during quiet standing, a normalized measure of the initial H reflex size (i.e., H reflex at 0 TA EMG). The box-whisker plots in Fig. 3*A* illustrate this finding ($P = 0.29$, Mann-Witney *U* test; $P = 0.43$, Wald-Wolfowitz runs test). The example in Fig. 4 shows that the relation between the H reflex and the TA EMG was the same during the tonic voluntary task and during leaning backward—a postural maintenance task.

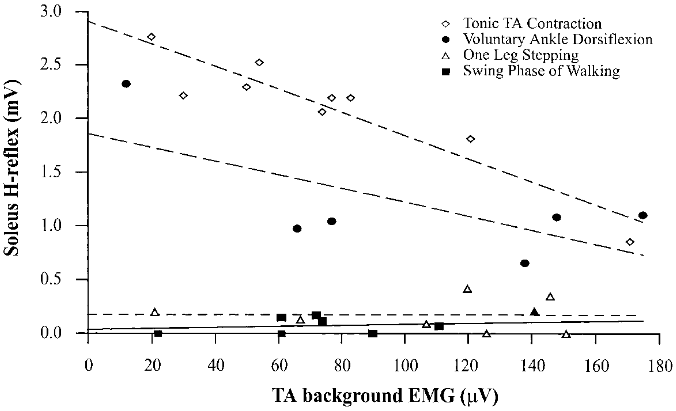

FIG. 2. Amplitude of soleus H reflex plotted against mean value of TA electromyographic (EMG) activity in the 4 motor tasks that were investigated in this subject. Results are from a single experimental session. It is clear that each task has a characteristic relation between these 2 variables. During swing phase of walking, or in 1-leg standing (OLS) task, the H reflex is essentially completely inhibited. During a voluntary tonic contraction of TA, H reflex is inversely related to TA EMG. In the voluntary ankle dorsiflexion task (target tracking), H reflex amplitude is smaller than in the tonic contraction task, but larger than during walking or OLS. *Y*-intercept value in tonic TA contraction task was 51% of maximum M wave (M_{max}).

In marked contrast, the soleus H reflex was very strongly inhibited during the swing phase of walking (Figs. 2 and 5). It is clear from Figs. 2 and 5 that at equal levels of TA EMG activity the H reflex is much more strongly inhibited during walking than in the other tasks, except for the OLS task. The soleus H reflex attains its minimum value just before or at the onset of the TA EMG burst in the swing phase. In 24 of 31 cases the H reflex was completely inhibited during the swing phase of walking. In 7 of 31 cases the H reflex during the swing phase was reduced to on average 5% of H_{max} during quiet standing (range 1–9 ± 3.0%, mean ± SD). Subjects with a H_{max}/M_{max} ratio >60% were more likely to have a residual H reflex during swing than subjects with a smaller ratio (Fig. 3*B*). The H reflex was similarly inhibited during the OLS task (Figs. 2 and 5, *A* and *D*). The strong inhibition of the H reflex during the OLS task was not dependent on ankle movement. It occurred

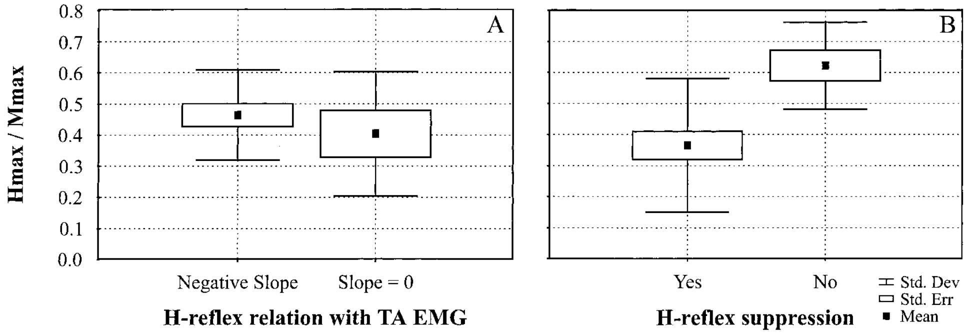

FIG. 3. Box-whisker graphs summarizing association between maximum H reflex (H_{max})/M_{max} ratio—a normalized measure of initial H reflex size—and whether or not H reflex decreased in proportion to TA EMG activity during voluntary tonic contractions (*A*) or whether or not H reflex was completely suppressed during swing phase of walking (*B*). There was no association between H_{max}/M_{max} ratio and whether or not H reflex decreased in proportion to EMG activity of TA during tonic contractions. On the other hand, it was more likely for H reflex to be completely inhibited during swing phase of walking, if H_{max}/M_{max} ratio was <60%.

with the same amplitude and time course when the foot was fixed in a rigid brace (Fig. 7*B*). The strength of the inhibition was also essentially independent of the strength of the TA EMG activity; for example, compare ''strong'' TA activation in Fig. 7*B* with the others. The reason for this is explained below.

When subjects produced a voluntary dorsiflexion of the ankle following the same trajectory as occurred during the swing phase of walking, the H reflex responses were of intermediate size compared with those of the tonic voluntary effort and those of the walking/OLS task (Figs. 2 and 5). Figure 6 shows examples comparing the EMG activity and ankle angular displacement during walking, OLS, and the target tracking task. Note the generally similar amplitude and velocity of the ankle dorsiflexion and the similarity of the TA EMG activity in all three tasks. The correlation between the SOL H reflex and the TA EMG was weak in the voluntary dorsiflexion task, as can be seen in the examples shown in Figs. 2 and 5. Of 10 subjects, 3 had a significant negative correlation between these two variables that accounted for ~68% of the total H reflex variance. The main reason for the generally poor correlation between these variables was that the inhibition of the H reflex was completed by the time of TA EMG onset (7 of 10 subjects).

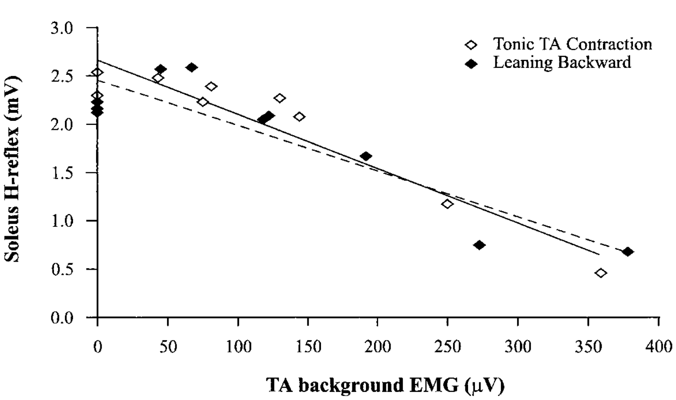

FIG. 4. Example from 1 subject of relation between soleus H reflex and TA EMG activity during voluntary tonic activity of TA compared with leaning backward. In the 4 subjects studied in these 2 tasks there was no statistical difference between fitted least-mean-squares lines. *Y*-intercept value in tonic TA contraction task was 83% of M_{max}.

The H reflex was also correlated with the kinematic variables of the voluntary dorsiflexion task. It should be noted that in this task H reflexes were elicited starting at the onset of TA EMG activity and throughout the movement until the final position was attained. Thus H reflexes occurring during the reaction time were not included in the analysis, because they would not be correlated, ipso facto, with either the EMG activity or movement-related variables. There was no correlation between the H reflex amplitude and the ankle angular velocity. In some subjects (4 of 10) there was a correlation between the H reflex amplitude and the ankle angular displacement, but this correlation was incidental and relatively weak, accounting for ~57% of the total H reflex variance. The incidental nature of the correlation is clear when considering that in two subjects the correlation was positive (i.e., the H reflex decreased with dorsiflexion), and in the other two the correlation was negative (i.e., the reflex increased with dorsiflexion). These correlations occurred because in some cases the H reflex was very small at the start of the ankle dorsiflexion and recovered to some extent at the end of the movement, or conversely in some cases it continued to decrease to a small extent after movement onset, as in Fig. 8. It is clear from the graphs in Fig. 8 that the inhibition began before TA EMG onset but that, in this example, the minimum H reflex value occurred at the peak of the EMG burst slightly after the beginning of ankle dorsiflexion. In summary, in 6 of 10 subjects there was no correlation between the size of the H reflex and ankle angular displacement or velocity, and in the other four subjects the correlation with the ankle angular displacement was incidental. The poor correlation of the H reflex amplitude with either EMG activity or movement-related variables suggests that it is principally determined by central neural mechanisms in anticipation of movement-related events, rather than as a consequence. Further evidence for a strong central control of reciprocal inhibition in the more complex OLS task is presented below.

Time course of the H reflex inhibition

In the OLS task the soleus H reflex attained its minimum value before movement of any leg joint in 11 of 12 subjects. Two examples of the behavior of the H reflex during the

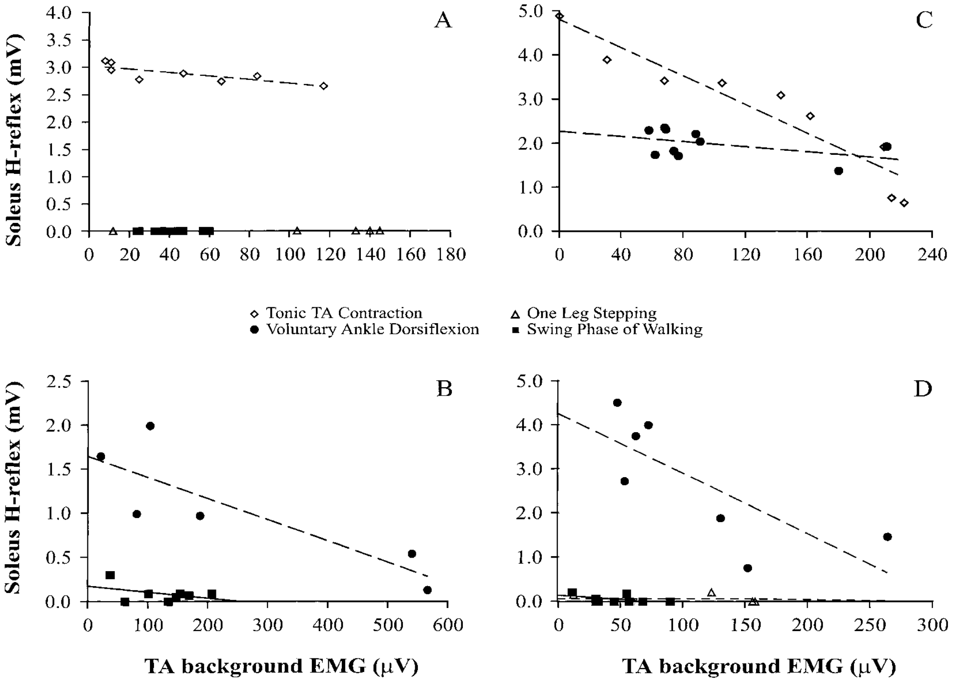

FIG. 5. Further examples of relation between soleus H reflex amplitude and TA EMG activity in various tasks. Each graph is from a different subject studied in a single experimental session. Note again that during swing phase of walking and OLS, H reflex is essentially completely inhibited. Note also that during voluntary ankle dorsiflexion (target tracking), in which correlation between plotted variables is poor or nil, H reflex amplitude is smaller than in tonic voluntary contraction task (e.g., Fig. 5C), but larger than during swing phase of walking or OLS.

OLS task are shown in Fig. 7. The arrows under the time axis indicate the onset of the EMG activity in TA—the first muscle activated in this task. It is clear that the strong inhibition of the H reflex is essentially completed during the reaction time. In four subjects the H reflex attained its minimum before TA EMG onset; in the other seven it reached its minimum before ankle movement. In only one subject did the soleus H reflex reach its minimum after movement initiation. Nonetheless, even in this subject, the inhibition began some 145 ms before movement onset and 77 ms before TA EMG activity. For the group as a whole, the H reflex began to decrease on average 122 ± 42.2 (SD) ms ($n = 12$) after the auditory go signal and 44 ± 30.1 (SD) ms ($n = 12$) before the onset of TA EMG.

DISCUSSION

Two main new findings concerning reciprocal inhibition of antagonist muscles have been described. First, reciprocal inhibition of the soleus is very much stronger during the swing phase of walking than it is during voluntary or postural tasks at matched levels of TA EMG activity. This demonstrates that the strength of reciprocal inhibition can be controlled independently of the level of motor activity of the agonist(s). Second, within a given task the strength of the inhibition is not necessarily proportional to the level of motor activity in the agonist(s). Additionally, on the basis of the measurements of the H reflex amplitude during the reaction time of voluntarily initiated single-joint or complex coordinated multijoint movements, it is clear that the inhibition is for the most part centrally mediated. This result is consistent with and extends what has been reported for isometric contractions at a single joint (Crone et al. 1987; Gottlieb et al. 1970; Kots 1969).

These findings further our understanding of reciprocal inhibition in several respects. Reciprocal inhibition is stronger in tasks involving joint movement than during tonic voluntary activity or postural maintenance. This likely reflects the need to strongly suppress the powerful stretch reflexes of the ankle extensors during rapid dorsiflexion movements (Capaday and Stein 1986; Gottlieb et al. 1970; Stein and Capaday 1988). However, additional factors related to the task itself are also involved, because, for example, the inhibition is much stronger during walking and OLS than it is when only the ankle is voluntarily dorsiflexed. Closely related to this is the idea that the differences in the strength of reciprocal inhibition occur in anticipation of movement-related events rather than as a consequence. The greater strength of the reciprocal inhibition during walking and OLS may also explain why the H reflex reached its minimum value before TA EMG onset in 90% of cases, compared with 70% in the voluntary ankle dorsiflexion task.

Below, the rationale and limitations of using the mean value of the surface EMG as a measure of the activity of a motoneuron pool are presented. This is followed by a discussion of the central versus peripheral origin of the task dependence of reciprocal inhibition, the possible neural mechanisms involved, and the functional implications of the findings.

Surface EMG as a measure of the activity of a motoneuron pool

The mean value of the rectified and filtered EMG is related to the number of active motoneurons and their discharge rate (De Luca 1979; Milner-Brown and Stein 1975). As long as motor units increase their discharge rate in parallel with the EMG and are recruited in the same order in the different tasks investigated (Desmedt and Godaux 1978; Grimby 1984; Hoffer et al. 1987; Milner-Brown et al. 1973), the mean value of the rectified EMG will be a fair measure of the level of activity of the motor pool. In the tasks investi-

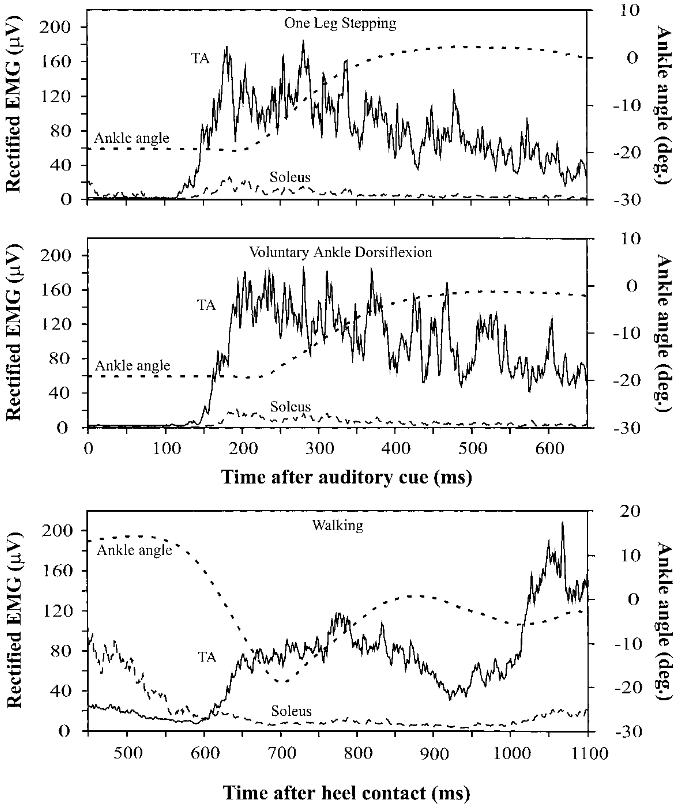

FIG. 6. Example showing EMG activity of TA and soleus during OLS, voluntary ankle dorsiflexion (target tracking), and walking in 1 subject. Ankle angular displacement is also shown for each task. Upward deflections of displacement trace: ankle dorsiflexion. Note similarities of EMG amplitudes and profiles, as well as similar amplitude of ankle dorsiflexion in each task. Walking EMG appears smoother and thus smaller in amplitude because it is the average of more trials ($n = 32$) compared with the EMG traces of the other 2 tasks ($n = 8$). Mean value of TA EMG between 644 and 944 ms during walking was 83 ± 31 (SD) μV Over an equal time interval, mean value of TA EMG was 89 ± 34 (SD) μV in OLS task and 114 ± 35 (SD) μV during voluntary ankle dorsiflexion.

gated in the present study, because they involved contractions of moderate speed, or graded nearly isometric contractions, the recruitment order was likely to be similar (Jakobssen et al. 1988). However, in comparing tasks in which the degree of synchronization of motor unit discharge may be widely different, such as ballistic contractions compared with more slowly graded contractions, the same mean value of the rectified EMG may not represent the same combination of active motor units and discharge rate. In conclusion, within these limits, matching EMG levels across tasks is a minimum requirement for demonstrating a task-dependent and nontrivial change in the input-output parameters of a neural pathway (Capaday 1997; Devanne et al. 1997).

Origin of the task dependence of reciprocal inhibition

Several observations strongly support the idea that the observed task-dependent differences in the strength of reciprocal inhibition are due, for the most part, to central factors. First, in tasks involving a reaction time the soleus H reflex attains its minimum value, in most cases, before the onset of EMG activity or joint movement. Similarly, during the swing phase of walking the H reflex attains its minimum value just before, or at the onset, of TA EMG activity. The similar behavior of the H reflex during walking and the OLS task also strongly suggests that a substantial part of the inhibition during the swing phase of walking is centrally determined. This is consistent with the conclusions of Whelan and Yang (1993), based on a different experimental paradigm. On the other hand, it has been suggested that during cycling and walking, movement-related afferent activity (reafference) from knee extensor afferents has an inhibitory action on the H reflex (McIlroy et al. 1992; Misiaszek et al. 1995). This is clearly not a contributing factor in the voluntary motor tasks investigated in this study for the reasons given above. To what extent reafference from the knee extensors contributes to the inhibition of the H reflex during walking cannot be determined with certainty from our measurements. However, we emphasize that the inhibition of the soleus H reflex during the swing phase of walking is very closely associated with the onset of TA EMG activity. Furthermore, the minimum value of the H reflex occurs when the knee is just beginning to flex and well before it reaches maximum flexion (Lavoie and Capaday 1996; Winter 1991). We suggest, therefore, that the inhibition of the so-

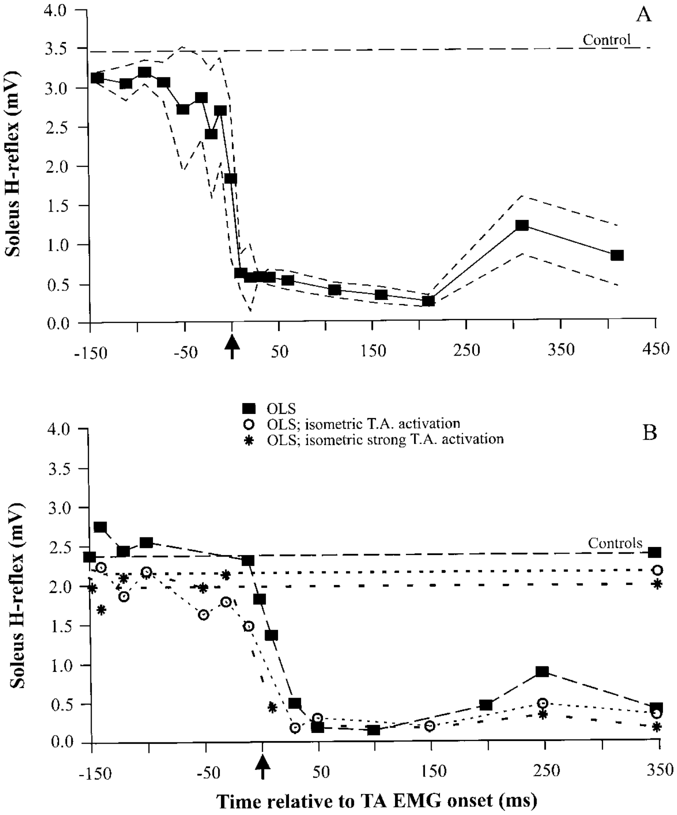

FIG. 7. Time course of inhibition of H reflex during OLS tasks. *A* and *B* are from 2 different subjects. Arrow beneath time axis of each graph: onset of TA EMG activity. Data points to *left* of arrow were obtained during reaction time, i.e., before any EMG activity or movement of any joint. Note how H reflex reaches its minimum near onset of TA EMG activity. Small recovery of H reflex some 200 ms after onset of TA EMG activity occurs at end of ankle dorsiflexion, when the ankle reaches its final position (Figs. 6 and 8). Dashed lines around time course curve in *A*: mean ± SD. Mean value of control H reflex was obtained by stimuli applied simultaneously with the auditory "go" signal. Control values: 89% of $M_{\max}$ (*A*); 65%, 61%, and 54% of $M_{\max}$ (*B*).

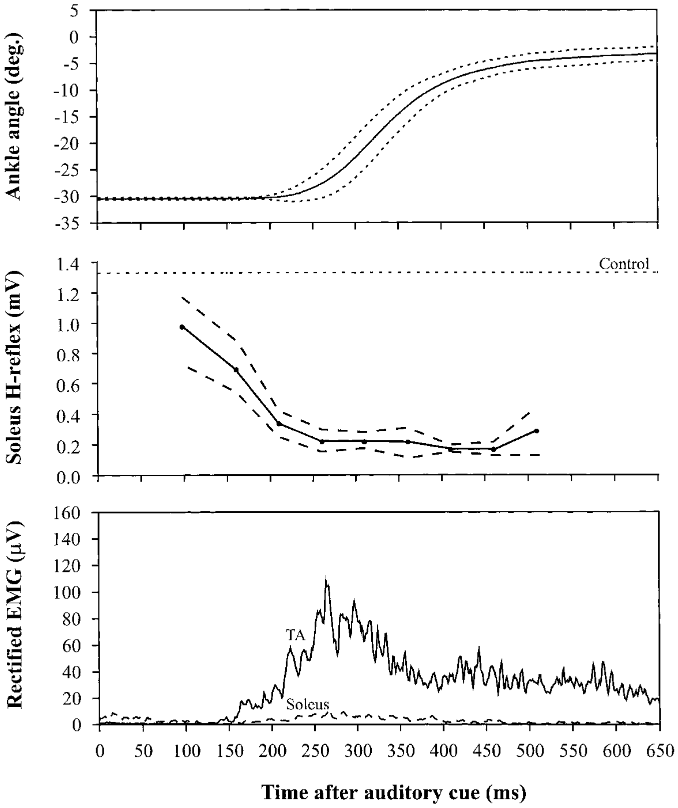

FIG. 8. Example of time course of soleus H reflex during voluntary dorsiflexion involving target tracking as described in text. Mean value of control H reflex, 46% of $M_{\max}$ in this example, was obtained by stimuli applied simultaneously with the auditory go signal. In all cases, H reflex began to decrease before onset of TA EMG activity. In some subjects, as in example shown here, it had not reached its minimum value by the time of movement onset, although it did so very soon thereafter. The point of this figure is that in such cases it is possible to obtain a correlation between H reflex amplitude and ankle angle, but as explained in the text, this correlation is incidental. Dashed lines: mean ± SD.

leus H reflex during the swing phase of walking follows the classic pattern of reciprocal inhibition between antagonist muscles and that it is therefore, for the most part, centrally determined. Indeed, recent work has shown that reciprocal inhibition of ankle extensors, including the disynaptic component, occurs despite the fact that the flexor nerve is anesthetized, clearly demonstrating that the process is centrally determined and that afferent activity (e.g., via the gamma loop) is not required (Nielsen et al. 1995; Sinkjaer et al. 1995). It is possible, however, that reafferent activity may serve to further ''clamp down'' the H reflex in proportion to the kinematic parameters. In particular, postactivation depression of synaptic transmission from Ia afferents to α-motoneurons (Hultborn et al. 1996) is a potential mechanism—initiated by muscle stretch—that may contribute to the clampdown of the H reflex.

Can possible differences in the task-dependent recruitment of different muscles account for the results? It may be suggested, for example, that the extensor digitorium longus and the peroneous longus are inactive during tonic contractions, but recruited during walking. EMG recordings showed that this was not the case, although quantitative comparisons between tasks were not made because it is difficult to record from these muscles free of cross talk. Regardless of possible small differences in muscle synergies between tasks, we suggest that any effects on the inhibition of the soleus H reflex are determined by the CNS acting in a permissive manner. In summary, the inhibition of the H reflex is centrally determined in anticipation of the movement, but reafferent signals may provide an additional contribution to ensure that the reflex remains strongly inhibited.

Possible neural mechanisms

There are several possible neural mechanisms that can control the strength of reciprocal inhibition of antagonists independently of the strength of the excitatory drive to the agonist(s). Figure 9 illustrates the principal neural circuits that may be involved. It may be suggested, for example, that the strong inhibition of the soleus H reflex during the swing phase of walking is due to increased hyperpolarization of the soleus motoneurons and an increase of presynaptic inhibition of their Ia-afferent terminals (Fig. 9). A descending pathway with a greater density of projections to the IaIns would produce a greater inhibition of the antagonist motoneurons at equal levels of excitatory drive to the agonist motoneuron pool. Thus one may speculate that during voluntary activity the corticospinal tract determines the input-output properties of the reciprocal inhibitory pathway, whereas during walking the spinal motor apparatus may be driven, in whole or in part, by other descending systems having more potent connections with the IaIns and presynaptic inhibitory interneurons. Control of recurrent inhibition of the IaIns by the Renshaw cells is another possible site at which the strength of reciprocal inhibition may be modified (Fig. 9). Mutual inhibition between IaIns (Baldissera et al. 1981) belonging to antagonist motoneuron pools is yet another possible site of action. Finally, it is conceivable that other inhibitory pathways activated by flexor nerves, such as the long-latency inhibition described by Crone and Nielsen (1989), or the one described by Capaday et al. (1995), may be involved. It is clear that future work is required to identify the neural circuits involved and what adaptive advantage each contributes.

Functional implications

Several important principles on the operation of reciprocal inhibition have been shown in the present study. In most subjects during tonic activity—whether generated voluntarily or as a result of postural requirements—the amount of inhibition to the antagonists increases in proportion to the level of motor activity in the agonist(s). This is in agreement with the hypothesis of Lundberg (1970) on the operation of the disynaptic inhibitory pathway. However, it is clear that in some subjects this is not the case; the antagonist(s) is little inhibited during activity of the agonist(s), except at very high levels of agonist activity, as determined by the lack of correlation between the soleus H reflex amplitude and the level of TA EMG activity. This has been reported previously by El Tohamy and Sedgwick (1983), who argued that their finding was contrary to the proportional decrease reported by Gottlieb et al. (1970). The present study recon-

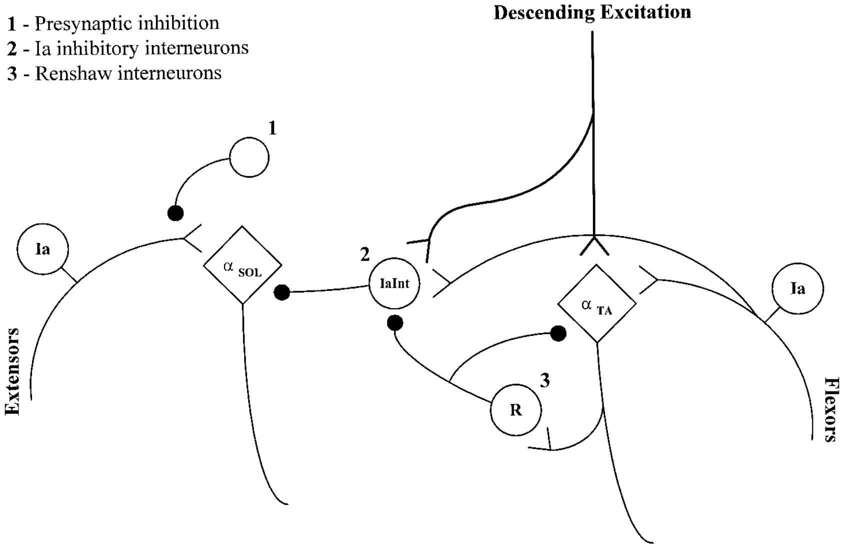

FIG. 9. Schematic representation of spinal cord neural circuits most likely involved in controlling strength of reciprocal inhibition of soleus α-motoneurons. Three main sites of action may be involved. An increase of strength of reciprocal inhibition, defined functionally, can occur when increasing presynaptic inhibition (*1*) of Ia-afferent terminals projecting to soleus α-motoneurons is increased. A descending pathway with more potent connections with Ia-inhibitory interneurons (IaIns, *2*) would also produce a greater reciprocal inhibition of soleus α-motoneurons at matched levels of recruitment of TA motoneuron pool. Finally, a reduction of recurrent Renshaw inhibition (*3*) of IaIns may also enhance reciprocal inhibition of soleus motoneuron pool. Further details are given in text.

ciles these rival positions; both types of behavior occur, the proportional decrease being the most common. Furthermore, it was shown (Fig. 3) that this did not depend on the initial size of the H reflex (i.e., when the TA is not active). Thus the principle that emerges is that the extent to which inhibition of the antagonist(s) is coupled to activation of the agonist(s) may not be the same in different subjects. The reason why this observation may be important is based on the predictions of a computer model of the input-output properties of motoneuron pools (Capaday 1997). If the reciprocal inhibition was produced only by hyperpolarization of the soleus α-motoneurons, the model predicts that the steepness of the relation between soleus H reflex amplitude and the level of TA EMG activity should depend on the size of the initial H reflex. This may therefore be a hint that other mechanisms may be involved, such as presynaptic inhibition of the soleus Ia-afferent terminals, in addition to reciprocal disynaptic inhibition (Crone and Nielsen 1989; Fu et al. 1978; Nielsen and Kagamihara 1993).

Why is the soleus H reflex not completely inhibited during tonic flexor activity? During tonic flexor activity the ankle extensors are not stretched, in the dynamic sense, and thus their stretch reflex would not oppose flexor activity. There is thus no need to strongly suppress the stretch reflex of the extensors, and the observation may simply be an example of the ''economy'' of neural activity. On the other hand, maintenance of some level of excitability in the segmental circuits of the antagonists may allow for their rapid recruitment when necessary, such as in the maintenance of equilibrium during postural tasks.

A second principle of operation is that the strength of inhibition is greater in tasks involving joint movement. However, the poor correlation of the H reflex with either EMG activity or movement-related variables, in addition to the fact that the H reflex reaches its minimum essentially during the reaction time, suggest that the strength of reciprocal inhibition is principally determined by central neural mechanisms in anticipation of movement-related events, rather than as a consequence. Indeed, it would be surprising if the CNS did not incorporate into the motor commands some degree of anticipatory control of reflexes that would oppose the task. Elements of this idea go at least as far back as the work of Kots (1969) and Gottlieb et al. (1970).

A third principle is that in addition to joint movement, per se, there must be a contribution from the task itself. Thus the uniquely strong inhibition of the soleus H reflex during the swing phase of walking and the closely related OLS task can be understood in terms of the motor control requirements in this part of the gait cycle (Capaday and Stein 1986). During the swing phase the foot moves forward only a few millimeters above the ground and the body is balanced on one leg. The forces that need to be dealt with for controlling the foot trajectory include inertial, viscous, and joint interaction forces. Any erroneous control action at this time may lead to stumbling, or injury, if the foot were to hit the ground. Thus the strong suppression of the powerful extensor stretch reflexes may be part of the ensemble of neural mechanisms to ensure foot clearance.

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