On the Origin of the Soleus H-Reflex Modulation Pattern During Human Walking and Its Task-Dependent Differences

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Schneider, Cyril, Brigitte A. Lavoie, and Charles Capaday. On the origin of the soleus H-reflex modulation pattern during human walking and its task-dependent differences. *J. Neurophysiol.* 83: 2881–2890, 2000. Recently, Brooke and colleagues have suggested "that the strong inhibition arising from passive movement about the knee and hip joints, lays down the base for the soleus H-reflex gain modulation seen during human gait." In particular stretch-evoked afferent activity from the quadriceps muscle was emphasized as the most important source of movement-induced inhibition of the H-reflex. To test this hypothesis we examined the kinematics and electromyographic (EMG) activity of the leg during human walking and correlated these with the modulation pattern of the soleus H-reflex. To further test the possible contribution of stretch-evoked quadriceps afferent activity to the soleus H-reflex modulation pattern during walking different walking gaits were studied. In one condition subjects were asked to walk with their knee locked in full extension by a rigid knee brace. In a second condition subjects were asked to walk backwards. During normal walking, the soleus H-reflex modulation pattern is strongly correlated with the EMG events of the soleus and tibialis anterior (TA), but not with hip, knee, or ankle angular displacement or velocity. When subjects walked with the knee locked in full extension, the amplitude of the H-reflex, its modulation pattern, and the task-dependent changes of its amplitude were the same as during normal walking. During backward walking, the H-reflex increases in late swing before activity of the soleus has begun and while the knee is flexing, an observation that highlights central control of the H-reflex amplitude. The effects of imposed flexion of the knee in passive subjects were also reexamined. The knee flexion imposed by the experimenter followed the same trajectory as that which occurred during the swing phase of the subject's step cycle. It was found that imposed knee flexions elicited a burst of TA EMG activity with an average latency of 81.6 ms (SD = 21 ms) in six out of eight subjects. Inhibition of the H-reflex, when it occurred, was associated with the occurrence of this burst. When subjects voluntarily flexed their right knee from an initial quiet standing posture, the inhibition of the soleus H-reflex began before flexion of the knee or that of any other leg segment. Once again the onset of inhibition was closely associated with the onset of activity in the TA. In the discussion section the present observations are examined in light of the predictions made by the movement-induced inhibition hypothesis of Brooke et al. It will be concluded that none of the predictions of this hypothesis were corroborated by present tests done during human walking. In consequence, we suggest that the modulation pattern of the H-reflex observed during normal human walking is centrally determined, as are the task-dependent differences of its amplitude (e.g., standing versus the stance phase of human walking).

INTRODUCTION

The amplitude of the soleus H-reflex is strongly modulated during human walking and running (Capaday and Stein 1986, 1987a). It increases during the stance phase in a ramplike fashion in parallel with the soleus electromyograph (EMG) and is strongly suppressed, or completely inhibited, during the swing phase when the ankle flexor tibialis anterior (TA) is active. The following neural mechanisms contribute to the modulation of the soleus H-reflex during the normal step cycle: the increasing activity of the α-motoneurons during the stance phase (Capaday and Stein 1986), the increase of postsynaptic inhibition of the α-motoneurons during the swing phase (Lavoie et al. 1997; Pratt and Jordan 1987), and as previously suggested (Capaday and Stein 1987a,b), a tonic increase of presynaptic inhibition during the step cycle.

However, the relation between the amplitude of the soleus H-reflex and the background level of motor activity is strongly dependent on the motor task (Capaday and Stein 1986, 1987b; Edamura et al. 1991; Llwellyn et al. 1990; Morin et al. 1982). For example, at matched levels of EMG activity the H-reflex is much higher during standing than in the early part of the stance phase of walking (Capaday and Stein 1986; Morin et al. 1982). The task-dependent changes of the input-output properties of this neural circuit have been suggested to reflect an adaptive control of the stretch reflex parameters in accordance with the biomechanical exigencies of the motor task (Capaday 1995; Stein and Capaday 1988). On the basis of computer simulations (Capaday and Stein 1987b; see also Heckman 1994) and experimental studies in decerebrate cats (Capaday and Stein 1989), we suggested that the task-dependent changes of the input-output properties of this neural circuit were due to changes of presynaptic inhibition of the Ia afferents. The direct demonstration of task-dependent changes of presynaptic inhibition during posture and gait has been more difficult (Capaday et al. 1995; Faist et al. 1996). In any case, the locus and origin of whatever mechanism is involved in the task-dependent control of the H-reflex amplitude was never explicitly stated, although it was tacitly assumed that it was of central origin. Recently, however, Brooke et al. suggested that ". . . the strong inhibition arising from passive movement, about the knee and hip joints, lays down a base for the soleus H-reflex gain modulation seen during human gait" (Brooke et al. 1995). In particular stretch-evoked afferent activity from the quadriceps muscle was emphasized as the most important source of movement-induced inhibition of the H-reflex. The experimental evidence for this hypothesis was obtained from "passive" rotations of the whole leg or individual leg segments of human subjects (Brooke et al. 1995), or rotations about the knee joint in anesthetized dogs (Misiaszek et al. 1995). Brooke et al. (1995) emphasized that "the movement about the knee is a more powerful source of inhibition than movement about the

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hip" and "the substantial inhibition seen from movement about both the hip and knee joints may function to ensure that this powerful stretch reflex activation is suppressed during the swing phase, when it could seriously disturb the normal stepping and walking patterns." What is implied in this statement is that the inhibition of the H-reflex during the swing phase of walking is due to the knee flexion during the swing phase. However, this idea does not seem to have taken into account the central control reciprocal inhibition between antagonistic muscles (e.g., see Lavoie et al. 1997).

The movement-induced inhibition hypothesis of Brooke et al. (1995) involves a tonic inhibition and a phasic modulation. Thus one implication of this hypothesis is that there should be a close correlation between the kinematic parameters of the hip and particularly the knee and the modulation pattern and amplitude of the soleus H-reflex (phasic modulation). A second implication of the hypothesis is that movement-induced afferent discharge will have a general suppressive effect on the H-reflex throughout the cycle (tonic inhibition). None of the experimental data underlying the "movement-induced inhibition" hypothesis were obtained during human walking. Therefore, the purpose of the present series of experiments was to test the predictions of this hypothesis during human walking. The hypothesis clearly predicts the following outcomes. First, the modulation pattern of the soleus H-reflex should be closely correlated to the kinematics of the leg joints, especially that of the knee. Second, the kinematic events should precede the onset of the predicted changes in H-reflex amplitude, such as its inhibition during swing. Third, any change in the pattern of peripheral feedback, especially from the knee, should modify the H-reflex modulation pattern and the task-dependent changes of its amplitude.

We therefore tested in detail and with improved methods (Capaday et al. 1995; Lavoie et al. 1997) the predictions of this hypothesis and reexamined the alleged effects of passive flexion of the knee on the soleus H-reflex. First, the modulation pattern of the soleus H-reflex during walking was measured and correlated with kinematic variables of the leg joints and with the EMG activity of the soleus and TA. The same measurements were done when the subjects walked with a rigid knee brace thereby essentially eliminating movements about the knee. The H-reflex and the joint kinematics were also measured during backward walking. The idea was to study the influence of a modified pattern of locomotor knee movements on the soleus H-reflex. Finally, we reexamined the effects of knee flexion imposed by the experimenter on the soleus H-reflex, as well as voluntarily initiated knee flexions. Part of this work has been reported in an abstract (Lavoie and Capaday 1996).

METHODS

The experiments were done on 24 normal human subjects ranging in age between 23 and 42 yr (mean = 28.3, SD = 6.3). All subjects gave their consent after being informed of the nature and purpose of the experiment, which was approved by the local ethics committee. The modulation pattern of the soleus H-reflex during walking on a treadmill was determined with improved methods as described below (real-time control of M-wave amplitude, background EMG, etc.). The H-reflex modulation pattern was also determined in seven subjects who walked on the treadmill with the knee of the experimental leg locked in full extension by a rigid knee brace (Orthèses Savard, Québec City, Canada). Six other subjects were asked to walk backwards at the same speed they had walked forward and the H-reflex modulation pattern was determined in each task. In the last series of experiments eight subjects stood with one leg on a stool while knee flexions were imposed by an experimenter on the experimental leg, which hung freely over the edge of the stool. The imposed knee flexions followed the same movement trajectory as occurred in the sagittal plane during the swing phase of normal walking. The soleus H-reflex was elicited before and at various times during the imposed knee flexions. For all subjects the data were obtained during a single experimental session.

Electromyographic recordings and stimulation

EMG recordings were obtained from the soleus, TA, vastus lateralis (VL) or rectus femoris (RF), biceps femoris, and gluteus maximus of the right leg with bipolar Ag-AgCl electrodes (7 mm in diameter) filled with saline gel and placed over the respective muscle bellies. H-reflexes were elicited by electrical stimuli of 0.5 ms duration to the tibial nerve in the popliteal fossa. The cathode was an Ag-AgCl electrode (7 mm in diameter) filled with saline gel and held in place by a double-sided adhesive ring collar and a rubber band wrapped around the leg. The anode consisted of a thin brass plate (3×7 cm) covered with gauze and moistened with saline. It was placed over the patella above the knee. A large ground plate was placed over the upper part of the calf, between the stimulating electrode and the recording electrodes. Electrogoniometers (Biometrics Ltd, Newport, Gwent, UK) were used to measure the ankle, knee, and hip angular displacements in the sagittal plane. A pressure-activated switch was placed inside the shoe and served as a marker for the onset of the step cycle and data acquisition. In the knee-free and knee-locked walking tasks the foot switch was placed under the heel. In the backward walking task the switch was placed under the metatarsal-phalangeal joint of the big toe.

Experimental procedures

Subjects walked on a treadmill at their preferred speed (on average 5.0 km/h). To determine the modulation pattern of the soleus H-reflex during the step cycle, H-reflexes were obtained in 50 ms increments starting from heel contact (HC). However, to improve the temporal resolution at the transition from stance to swing, the H-reflexes were elicited in 10 ms increments. Electrical stimuli to the tibial nerve were delivered with an interval varying between one to five steps, at random. Only one stimulus was delivered per step cycle. Eight H-reflexes were averaged at each tested phase of the step cycle. The amplitude of the H-reflex during quiet standing was determined several times throughout the experiment (e.g., before waking, in the midst of walking trials, and at the end of the experiment). The amplitude and shape of soleus M-wave served as a measure of the effective stimulus strength delivered to the tibial nerve (Capaday and Stein 1986). During the experiments, the M-wave was monitored by a time-amplitude window discriminator implemented in software (Capaday et al. 1995). Responses having an M-wave whose amplitude was outside a narrow time-amplitude window (e.g., ± 0.2 mV, between 10 and 15 ms) were rejected on-line. Consequently, the coefficient of variation of the averaged M-waves were typically around 10%. The background level of activity of the soleus and TA was estimated from the mean value of the rectified and filtered EMG over a 50 ms time interval prior to the stimulus. Further methodological details on recordings, stimulation, and comparison of responses in different tasks at matched levels of background EMG activity can be found in previous publications from this laboratory (e.g., Capaday 1987; Capaday et al. 1995; Lavoie et al. 1997).

For each subject that served in the imposed knee flexion experiments, the trajectory of the knee angular displacement during the swing phase of walking was extracted from averaged records of his

own step cycles. The movement trajectory was displayed on a computer monitor in direct view of the experimenter. The knee flexions imposed by the experimenter had to match this trajectory to within ±5°, otherwise the trial was rejected on-line (Lavoie et al. 1997). Thus in the imposed knee flexion experiments the stimulus-elicited M-wave and the knee movement trajectory were tightly controlled.

Data reduction

The EMG activities and joint angular displacements were averaged on-line from consecutive step cycles ($n = 64$ step cycles) in which no stimulation was applied and stored on computer disk for further analysis. From these averages, variables related to joint angular displacements and EMG activity were measured (Figs. 1, 2, and 3). The mean value of the EMG activity of the respective muscles was determined from these averages. It was calculated over a 60 ms time window centered on the time of occurrence of the H-reflex (not the time of stimulation) in that part of the step cycle. The kinematic and EMG variables were correlated with variables related to the modulation pattern of the soleus H-reflex, as will be explained in RESULTS. Changes in the direction of the angular displacements (e.g., from flexion to extension) of the leg joints were determined by differentiation of the displacement traces.

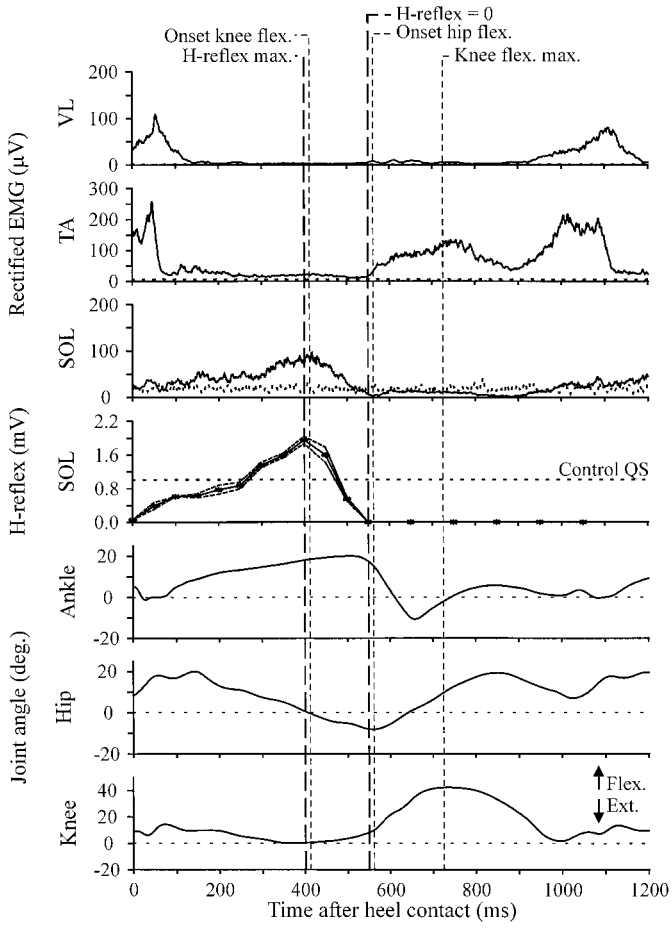

FIG. 1. Example from a single subject of the H-reflex modulation pattern during walking. Rectified electromyograph (EMG) activities of the vastus lateralis (VL), tibialis anterior (TA), and soleus are shown along with the corresponding soleus H-reflex amplitude (±SE) and the angular displacement of the ankle, hip, and knee. Data are plotted as a function of time after heel contact; step cycle duration is about 1100 ms. The H-reflex values are plotted against the time of their occurrence, not the time of stimulation in this and all other figures. The H-reflex amplitude during quiet standing is also indicated (control, QS). The rectified EMG traces are averages of 64 successive step cycles and the superimposed dashed traces are the EMG values during quiet standing. Joint flexion is shown as an upward deflection of the angular displacement trace. The thick dashed vertical lines are markers for the H-reflex maximum and the minimum value. The thin dashed vertical lines are markers of kinematics events: onset of knee and hip flexion, time of full knee flexion.

RESULTS

The results section is divided into five parts. In the first part we show that the soleus H-reflex modulation pattern during walking follows the classic pattern of centrally produced reciprocal inhibition between antagonistic muscles. That is, the H-reflex amplitude is highly correlated with the soleus EMG during the stance phase and the duration of its inhibition during swing is highly correlated with the duration of TA EMG activity. In contrast, in the second part we show that during walking the modulation pattern of the soleus H-reflex is not correlated with the kinematics of the leg segments. In the third section of the results we show that when subjects walk with the knee joint locked in full extension, the peak amplitude of the H-reflex does not increase in the stance or swing phase of the step cycle. Moreover, the task-dependent decrease of the H-reflex during the stance phase of walking compared with quiet standing occurs to the same extent in the knee locked and normal walking condition. In the fourth section we show that during backward walking there is a clear dissociation between the H-reflex modulation pattern and the knee movements. In the last section we show that in passive subjects imposed flexion of the knee elicits activity in the TA and that the inhibition of the soleus H-reflex, when it occurs, is tied to activation of the TA. Finally, we also show that when subjects voluntarily flex the knee, the soleus H-reflex is inhibited well before flexion of the knee or movement of any other leg segment.

Correlation between the soleus H-reflex and the agonist-antagonist EMG activities

Typical recordings of the EMG activity of the soleus, TA, and VL during the step cycle are shown in Fig. 1, along with the soleus H-reflex amplitude. It is readily apparent in Fig. 1 that the H-reflex amplitude is closely related to the EMG activity of the soleus and TA and shows little correlation with the leg joint kinematics. The H-reflex increases in a ramplike manner during the stance phase, in parallel with the soleus EMG activity. The maximal value is reached late in stance and drops suddenly as does the soleus EMG, before the onset of hip or knee flexion as indicated by the event markers in Fig. 1. The H-reflex reaches its minimum value (essentially zero in the 24 subjects studied) at the time the soleus EMG burst ends and just before the onset of the TA EMG activity. It remains at its minimum value throughout the swing phase, i.e., while the TA is active. At the time the H-reflex begins to decrease activity in quadriceps is negligible and the knee is just beginning to flex. Furthermore, hip flexion starts some 20–150 ms after the soleus H-reflex reaches its minimum value.

In Fig. 2, it is shown quantitatively that the modulation pattern of the soleus H-reflex during the step cycle is indeed strongly correlated with the duration of the soleus and TA motor bursts. The H-reflex reaches its maximum at nearly the same time as the peak of soleus EMG activity and reaches its minimum value at nearly the same time as the end of the soleus

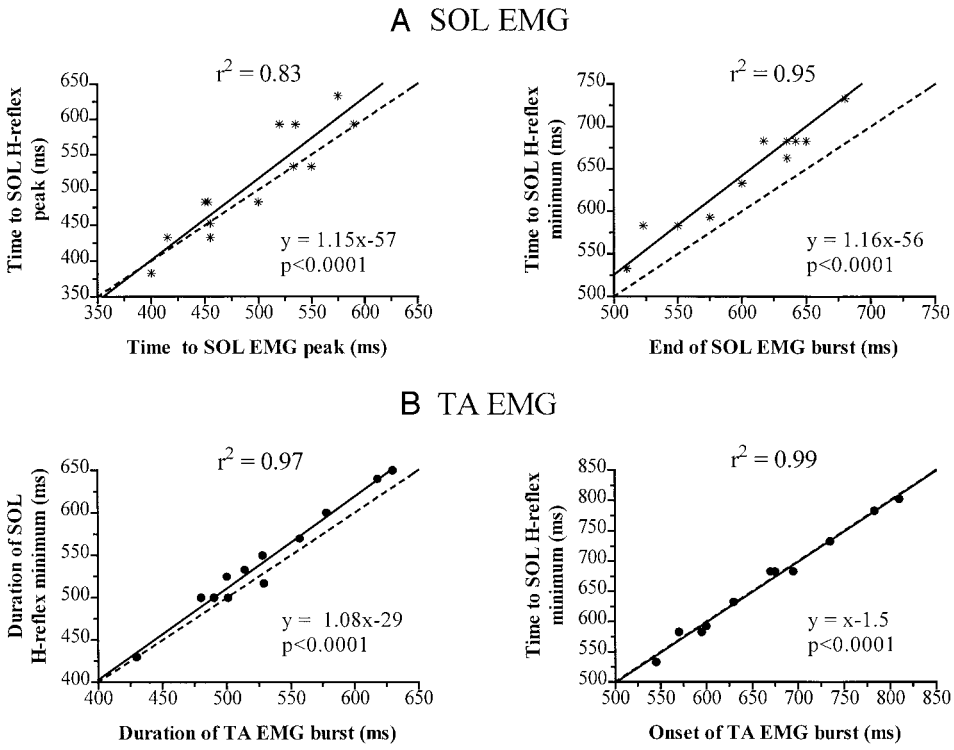

FIG. 2. Correlation between the soleus H-reflex and the EMG activity in the soleus (*A*) and TA (*B*) during the step cycle. Each point represents 1 subject and is the average of 8 values. The graphs in *A* show that the time to peak and the duration of the H-reflex above its minimum value are highly correlated with the equivalent parameters of the soleus EMG. The graphs in *B* show that the duration of the H-reflex inhibition during swing is highly correlated with the duration of the TA burst and that the onset of the inhibition occurs just before the onset of the TA burst. The correlation coefficient (r^2), its probability (p), and best fitting line parameters are given for each graph. Dashed lines in each graph has a slope of 1.

motor burst (Fig. 2*A*). The time at which the soleus H-reflex reaches its minimum value is also highly correlated with the onset of the TA EMG burst, occurring on average some 5 ms (SD = 2 ms) before the TA burst. The H-reflex remains at its minimum value during the swing phase for a time nearly equal to the duration of the TA motor burst (Fig. 2*B*).

In contrast, the modulation pattern of the soleus H-reflex during the step cycle is not correlated with the leg joint kinematics (Fig. 3). The focus of the analysis presented in Fig. 3 is on whether kinematic variables predict the onset and time of the H-reflex inhibition. It is clear that the parameters related to angular displacement or velocity of the leg joints are poorly correlated with the modulation pattern of the soleus H-reflex. The onset of the H-reflex decrease from its maximal value begins well before hip flexion and for the most part before the onset of knee flexion (Fig. 3, *A* and *B*). Note especially the lack of correlation between the onset of knee flexion and the onset of H-reflex decrease, or the time to reach the H-reflex minimum and the onset of knee flexion. The only significant correlation found was between the onset of ankle dorsiflexion and the time the H-reflex reaches its minimum (Fig. 3*C*). However, that correlation cannot be causal because the H-reflex time to minimum occurs on average 86 ms (SD = 18 ms) before ankle dorsiflexion.

Effects of blocking knee movements

The modulation pattern of the soleus H-reflex was quite similar in all seven subjects during walking with the knee locked (KL) in full extension (Fig. 4). In all cases, the soleus H-reflex amplitude was high during stance, dropped suddenly at the transition from stance to swing, and stayed at its minimum (essentially zero) throughout the swing phase. The soleus EMG burst during stance was similar in the KL task compared with normal walking, except for a small reduction in its duration and amplitude. The peak value of the soleus EMG was on average 116 μV (SD = 30 μV) during normal walking and slightly smaller at 100 μV (SD = 25 μV) in the KL task. The difference was not statistically significant (paired *t*-test, $P > 0.37$; $n = 7$ subjects). Closely related to this was the slight reduction of the peak H-reflex amplitude in the KL task compared with normal walking (4.6 mV, SD = 2.9 versus 5.2 mV, SD = 1.8). The difference, however, was not statistically significant (paired *t*-test, $P > 0.38$; $n = 7$ subjects). During the KL task the onset of the TA burst was clearly phase advanced in all subjects (mean = 41 ms, SD = 27 ms). The soleus H-reflex dropped to its minimum value just before the onset of the phase-advanced TA motor burst. The onset of the H-reflex inhibition during the KL task was thus closely associated with the onset of the TA burst, as in normal walking. This observation emphasizes the close association between the inhibition of the H-reflex and the onset of the TA motor burst. The reduction of the H-reflex during the early part of the stance phase of walking compared with quiet standing occurred to the same extent whether the knee was locked or free to move (Fig. 4). It should also be noted that the H-reflex reached its minimal value before hip flexion in both walking tasks.

In the knee-locked walking task movements at the hip and ankle are in fact noticeably reduced. All subjects ($n = 7$) produced the leg clearance in the swing phase by increasing the ankle plantar flexion on the contralateral leg, thus raising themselves higher than normal. Some subjects also used circumduction of the hip in addition (4/7). Changes in the EMG activity of the muscles in the KL leg did occur, as would be expected, but they varied from one subject to another. The important point is that they were not correlated with the soleus H-reflex modulation pattern. Focusing on the quadriceps activity in the KL condition we found that in 2/7 subjects the activity of the RF was phase advanced and increased in am-

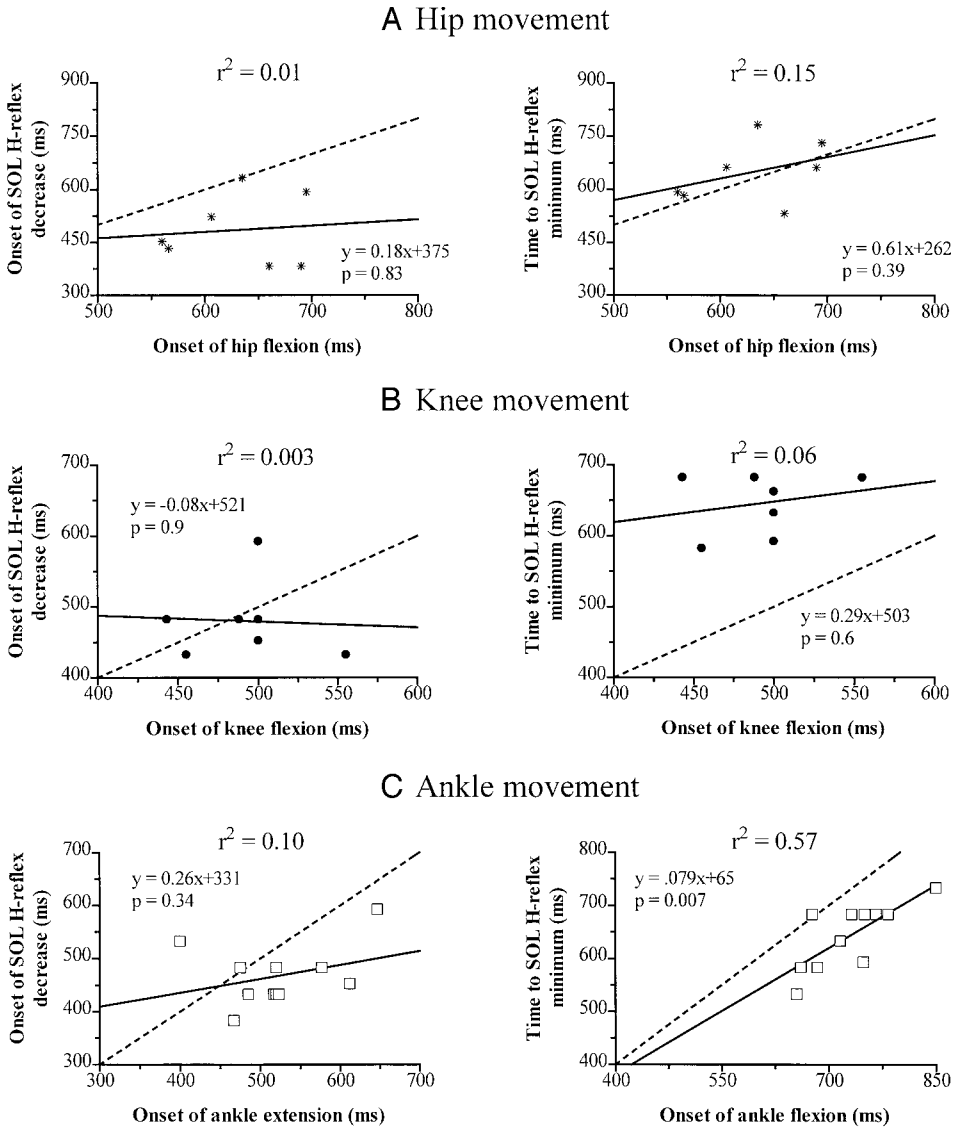

FIG. 3. Correlation of the soleus H-reflex with hip (*A*), knee (*B*), and ankle (*C*) kinematic events during the step cycle. Each point represents 1 subject and is the average of 64 values. The data show that neither the onset of the H-reflex decrease during stance nor the time to reach its minimum are correlated with flexion of the hip or knee. The only significant correlation was between the time to H-reflex minimum and ankle dorsiflexion, but this is not causal because the H-reflex minimum occurs before dorsiflexion (i.e., slope of the line <1). The correlation coefficient (r^2), its probability (p), and best fitting line parameters are given for each graph. The dashed lines in each graph has a slope of 1.

plitude by about 150% in 3/7 subjects. In two subjects RF activity did not change. In another two subjects there occurred a low-level tonic activity throughout the step cycle (about 45% of the normal locomotor bursts). The VL showed little activity in either task. Activity of the gluteus maximus was not markedly different in the two tasks.

Modulation pattern of the H-reflex during backward walking

The pattern of soleus H-reflex modulation was markedly changed when the subjects walked backwards (Fig. 5). The idea was to study the influence, if any, of a modified pattern of locomotor knee movements on the soleus H-reflex. In this task, the modulation pattern of the H-reflex is markedly out of phase with the soleus motor activity; its peak occurs after the peak of soleus EMG activity near toe contact. From that point, the H-reflex decreases as the activity in the TA increases during the remainder of the stance phase. At the onset of swing, the H-reflex drops to zero, just prior to a second burst of TA activity. In late swing a second peak occurs just before the onset of the soleus EMG burst, which serves to control lowering of the foot toward the ground. As can be seen in Fig. 5 during backward walking, the H-reflex is clearly not correlated with the knee angular displacement. More importantly, the increase of the H-reflex during late swing occurs while the knee is flexing and thus stretching the quadriceps.

Effects of imposed knee flexions on the soleus H-reflex

When the knee joint of relaxed subjects was rapidly flexed by an experimenter, the imposed knee flexion (IKF) elicited a burst of activity in the TA in six out of eight subjects (Fig. 6). The average latency of this TA burst was 81.6 ms (SD = 21 ms; range = 54–142 ms). The mean amplitude of this evoked burst was about 30% of the TA burst during the early swing phase of walking. In these subjects, the H-reflex is only inhibited in conjunction with activation of the TA (Fig. 6). In the subject whose data are shown in Fig. 6, when the ankle was braced at a joint angle of 90°, the burst of TA activity was eliminated and so too the inhibition of the H-reflex. In subjects in which IKF did not elicit TA activity, no inhibition of the H-reflex occurred (Fig. 7). This should be contrasted with what occurred during walking, where the H-reflex is nearly completely suppressed during the swing phase in close temporal

association with activity in the TA. The amplitude and time course of the knee flexion in walking is nearly the same as during the IKF task (Fig. 7). Another important point illustrated in Fig. 7 has to do with the shape of the M-wave during knee flexion. As can be seen in Fig. 7, not only is the M-wave amplitude nearly constant during knee flexion in both tasks, but so too is its waveform. In several subjects the M-wave shape changed substantially during IKF. In these subjects there was indeed an apparent reduction of the H-reflex. However, in such cases the cause of the reduction of the H-reflex is a moot point. It may be a consequence of elicited knee afferent activity or due to activation of a different fascicle(s) in the tibial nerve as a result of changes in the orientation and distance of the stimulating electrode.

Effects of voluntary knee flexions on the soleus H-reflex

When subjects voluntarily flexed their knee in response to an auditory signal, the soleus H-reflex was inhibited either during the reaction time or near the time of TA activation. In all cases the inhibition preceded movement of the knee or that of any other leg segment. An example of voluntarily initiated knee flexion starting from the quiet standing posture (i.e., both feet on the ground) is shown in Fig. 8. In this task, the TA is the first muscle activated and is followed by inhibition of the ongoing soleus EMG activity. The important observation is that the H-reflex inhibition begins near the onset of the TA EMG activity and well before movement of the knee.

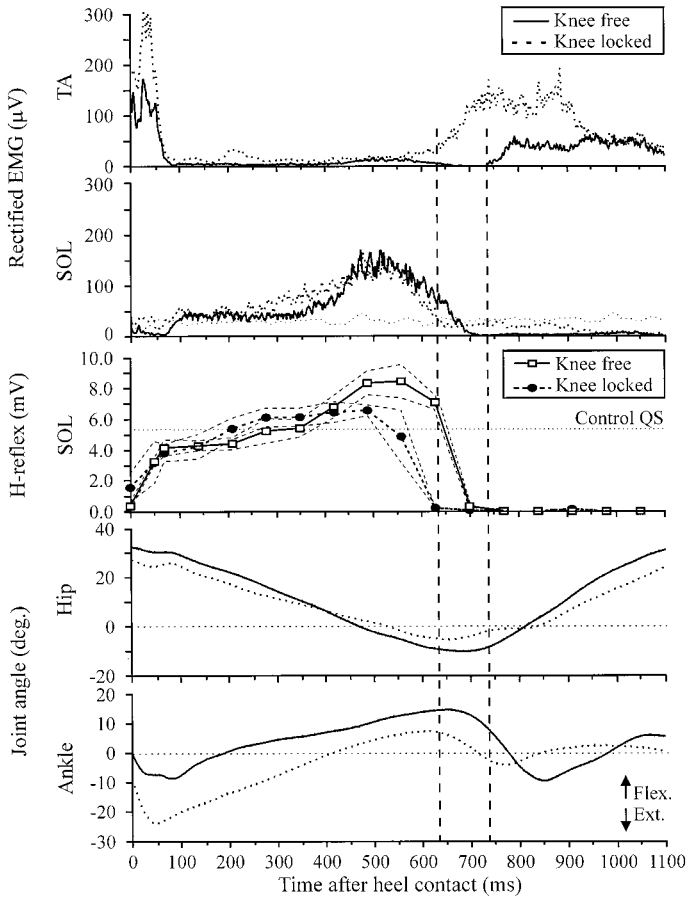

FIG. 4. Example from a single subject of the H-reflex modulation during walking with the knee locked in full extension and during normal walking. The rectified EMG of the rectus femoris (RF), TA, and soleus are plotted along with the corresponding soleus H-reflex amplitude (±SE) and the angular displacement of the hip and ankle. The data are plotted as a function of time after heel contact; the step cycle is about 1100 ms. The rectified EMG traces are averages of 64 successive step cycles during normal walking or walking with the knee locked at the same speed. The thin dotted soleus trace is the EMG activity during quiet standing. Amplitude of the H-reflex during quiet standing is also shown (control, QS). Note that the soleus EMG activity changes little during the knee-locked task. Note also the obvious phase advance of the TA in the knee-locked task and the corresponding phase advance of the H-reflex inhibition (vertical dashed line).

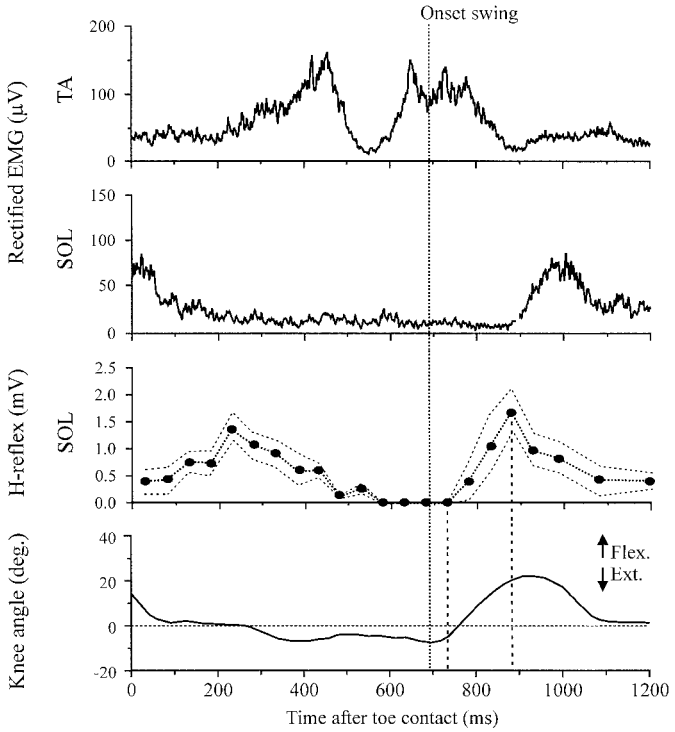

FIG. 5. Example from a single subject of the H-reflex modulation pattern during backward walking. The average (n = 64) EMG activity of the soleus and TA during the step cycle are shown along with the soleus H-reflex (±SE) and the knee angular displacement. The onset of swing is marked by the long dotted vertical line. Note the 2 peaks in the H-reflex modulation pattern during backward walking and the obvious lack of correlation with the angular displacement of the knee. Note also that in late swing the H-reflex increases while the knee is flexing (short dashed vertical lines).

DISCUSSION

In the discussion that follows the present observations are examined in detail in reference to the movement-induced inhibition hypothesis of Brooke et al. (1995) and in conjunction with our own past observations (Lavoie et al. 1997) and those from other laboratories (Garrett et al. 1999; Nielsen et al. 1995; Sinkjaer et al. 1995; Yang and Whelan 1993). The main point that will be made is that none of the predictions of the movement-induced inhibition hypothesis were corroborated during human walking. We conclude that movement-related activity has little effect on the amplitude or the pattern of the H-reflex modulation observed during human walking.

Modulation of the H-reflex by leg movements versus central control

We have shown that the duration of the soleus H-reflex during stance is highly correlated with the duration of the

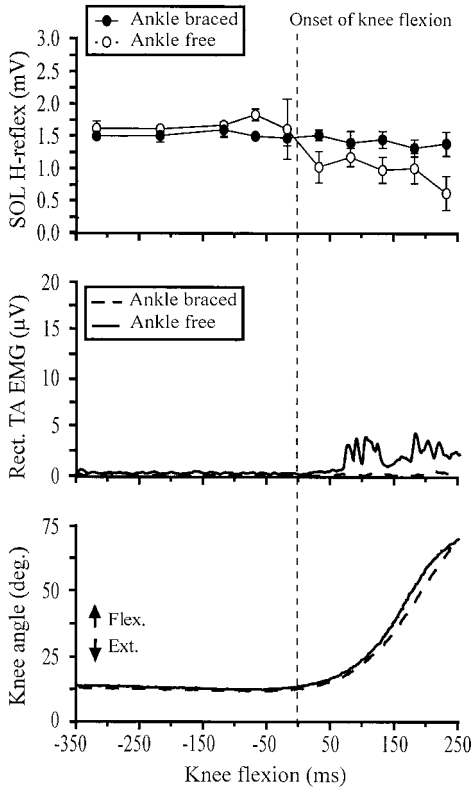

FIG. 6. Example of the effects of an imposed flexion of the knee (IKF) on the soleus H-reflex and on the EMG activity of the TA. Here an IKF, closely matched to the knee flexion angular displacement of the subject during walking, elicited a small EMG burst in the TA with a corresponding inhibition of the H-reflex. When the ankle joint was braced, thus preventing "foot drop" and reducing the need for activation of the TA, the evoked activity in the TA was nearly eliminated and so too the inhibition of the H-reflex.

soleus EMG activity. It was previously shown that its amplitude is also highly correlated with the soleus EMG activity (Capaday and Stein 1986). The duration of the H-reflex inhibition during the swing phase is highly correlated with the duration of the TA motor burst. This emphasizes that the modulation pattern observed during human walking follows the classic pattern of centrally produced reciprocal inhibition between antagonistic muscles (Jankowska 1992; Lundberg 1970). That is, the reflex is high during the stance phase when the soleus α-motoneurons are active and it is strongly inhibited during the swing phase when the antagonistic TA α-motoneurons are active. In marked contrast, we found no correlation between the modulation pattern of the H-reflex and the angular displacements, or velocities, of any leg joint. In particular, the onset of the H-reflex decrease in late stance, the time at which it reaches its minimum value at the stance to swing transition, and the duration of its inhibition during swing are not correlated with movements about the knee joint. The onset of H-reflex decrease occurs when the soleus EMG begins to decrease, not because of knee flexion. The time to reach the minimum value of the H-reflex at the stance to swing transition is highly correlated with the termination of the soleus motor burst and the onset of the TA motor burst (Figs. 1 and 2). Thus the idea that the H-reflex inhibition during swing is the result of afferent activity from the knee and hip seems untenable. Given the lack of correlation between the leg movement kinematics and the H-reflex modulation pattern, it is difficult to see how movement-related afferent activity can make an important contribution to the phasic modulation of the H-reflex.

Walking with the knee locked in extension

When subjects walked with a rigid brace which locked the knee in full extension the peak amplitude of the H-reflex during the stance phase did not increase, contrary to the prediction of the movement-induced inhibition hypothesis. The hypothesis predicts an increase of the H-reflex when knee flexion is blocked. What occurred was a slight reduction of the H-reflex associated with a slight reduction of the soleus EMG. This is a key observation because if knee movement is a critical variable for determining, or setting, the amplitude of the H-reflex during stance, the relationship between H-reflex amplitude and the level of motor activity should have been markedly affected; it was not. A small decrease of the EMG produced, as expected, a small decrease of the H-reflex (neither of which was statistically significant in our sample).

What is more, during the swing phase of the KL task the onset of H-reflex inhibition was highly and strikingly correlated with the onset of the phase-advanced TA EMG activity (Fig. 4). In other words, phase advance of the TA activity resulted in a phase advance of the H-reflex inhibition. Hip flexion could not have substituted for knee flexion because the H-reflex is completely inhibited prior to the onset of hip flexion. These observations suggest that the source of the inhibition is central and emphasize centrally produced reciprocal inhibition between antagonistic muscles as the mechanism underlying inhibition of the H-reflex during swing. In addition, the H-reflex was inhibited to the same extent in the swing phase in both walking conditions. If the inhibition in this phase of the step cycle depended on peripheral inputs from the knee mechanoreceptors, as suggested by Brooke et al. (1995), the H-reflex should have been larger during the swing phase of the KL task; it was not. This was also found independently by Garrett et al. (1999). In normal walking, flexion of the knee in early swing happens to roughly coincide with the onset of TA activity. We suggest that this has led to confusion of causal relationships.

Also contrary to the predictions of the movement-induced inhibition hypothesis, the reduction of the H-reflex during the early part of stance compared with quiet standing occurred to the same extent in the knee-locked condition compared with normal walking. This is also a key observation that makes two points at once. It is unlikely that the widely different pattern of afferent inputs to the CNS during the KL condition substituted exactly for the afferent inputs during normal walking to produce the same task-dependent change. From this it follows that the most likely explanation for the task-dependent differences in the input-output characteristics of this reflex is that it is centrally determined.

The same counterargument may be raised against all of our observations on modified locomotor patterns, viz. that the peripheral inputs to the CNS in the different conditions, indeed even a different pattern of γ-motoneuron activity, substituted exactly for the afferent input during normal walking. The ad hoc nature of these arguments is readily apparent (see also the discussion in Garrett et al. 1999).

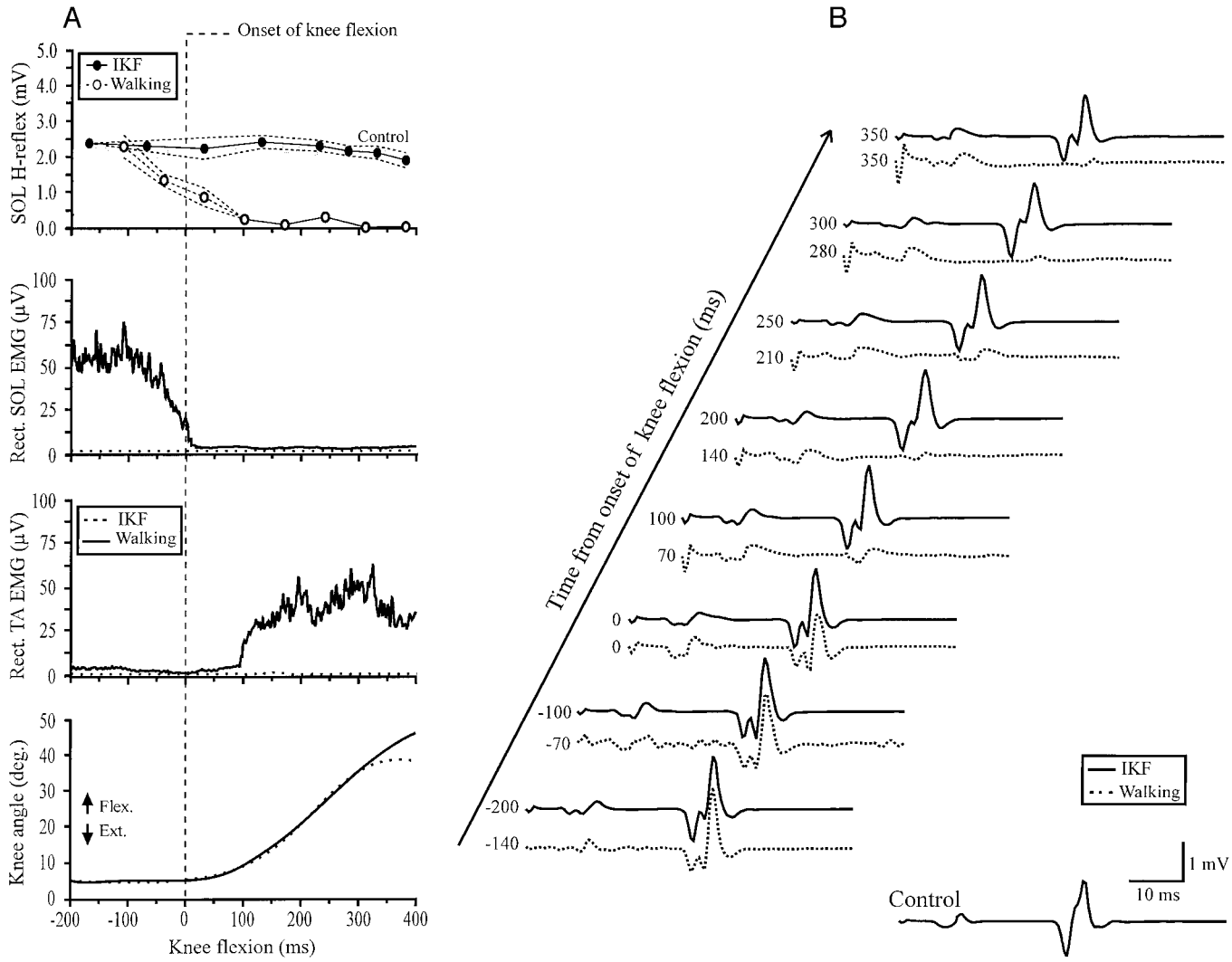

FIG. 7. Example in which IKF closely matched to the subject's knee angular displacement during walking did not elicit an EMG burst in the TA nor an inhibition of the soleus H-reflex (*A*). The control H-reflex (gray horizontal bar) is that obtained during quiet standing; the recording is shown as an inset to the figure in the lower right-hand corner. In contrast, during the swing phase of walking the H-reflex is inhibited in close conjunction with the TA burst. *B*: M-wave and H-reflex recordings measured at different times relative to the onset of knee flexion (used as an event marker) during walking or the IKF task. Note especially the constancy of M-wave shape and amplitude within a task and between tasks.

Neural mechanism of H-reflex inhibition during swing

Our suggestion that inhibition of the H-reflex during swing is due to central control of the reciprocal inhibitory pathway is entirely consistent with the findings of Nielsen et al. (1995), Sinkjaer et al. (1995), Yang and Whelan (1993) and our own observation on reciprocal inhibition during the human step cycle (Lavoie et al. 1997). Each of these studies concludes that the inhibition of the H-reflex during activation of the TA is centrally determined for the following reasons. It occurs whether or not there is peripheral feedback and is completed during the reaction time prior to activation of the TA. Nielsen et al. (1995) and Sinkjaer et al. (1995) have shown that during anesthetic block of the common peroneal nerve (thereby eliminating efferent and afferent activity) an intended activation of the TA inhibits the soleus H-reflex to the same extent as in the normal condition. In keeping with this, the soleus H-reflex is inhibited during the swing phase of walking even when the TA is voluntarily kept silent (Yang and Whelan 1993). Yang and Whelan (1993) went on to conclude that "specific feedback from the knee and hip of the ipsilateral side, at least, may not be essential in suppressing the soleus H-reflex." In our study of reciprocal inhibition during human walking and stepping (Lavoie et al. 1997), we showed that during a voluntarily initiated swing of the leg (akin to the swing phase of walking) the inhibition of the H-reflex is completed during the reaction time some 20 ms before activity in the TA and before movement of any leg joint. It is difficult to see what more peripheral feedback from the quadriceps, or elsewhere, can contribute.

Backward walking

When subjects walked backward, it was shown that during stance the H-reflex is out of phase with the soleus EMG activity and that its amplitude is reciprocally related to the TA motor activity (Fig. 5). This behavior is clearly not related to movement of the knee, or indeed the hip. The inverse relation between the soleus H-reflex and the TA activity during stance

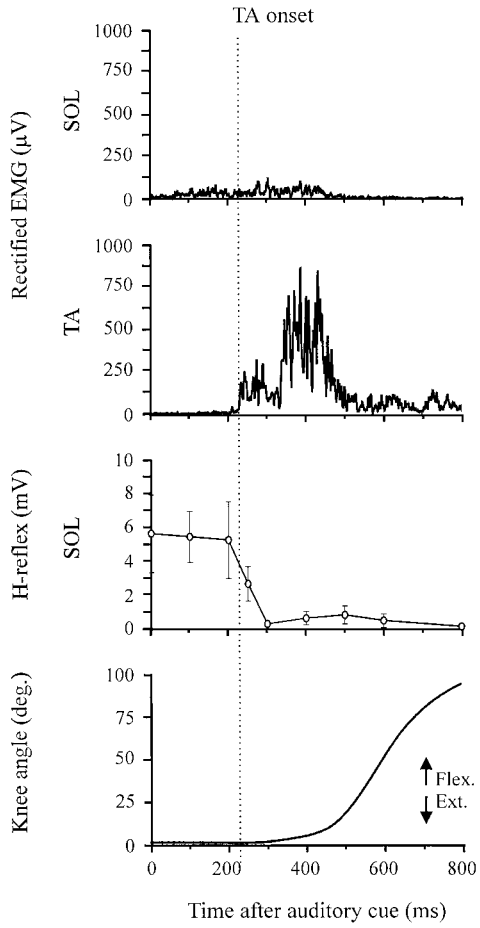

FIG. 8. During a voluntary knee flexion starting from the quiet standing posture the soleus H-reflex is inhibited during the reaction time of the movement. The subject was instructed to flex the knee in response to an auditory "go signal." In all subjects the H-reflex inhibition began during the reaction time prior to movement of the knee or that of any other leg segment. The time of maximum inhibition of the H-reflex occurred just before the onset of the TA motor burst.

again emphasizes the actions of reciprocal inhibition between antagonistic muscles. In late swing the soleus H-reflex increases just prior to the increase of soleus activity and toe contact. This increase occurs while the knee is flexing, thus stretching the quadriceps (the monoarticular heads at least). Proponents of the movement-induced inhibition hypothesis would argue that the H-reflex would be even greater if the knee did not flex. This is very difficult to understand because the H-reflex amplitude is greatest at a point in the step cycle when the soleus EMG is smallest. The most parsimonious interpretation of the complete lack of correlation between the H-reflex and knee flexion during backward walking is that its amplitude and modulation pattern are determined by central neural mechanisms.

Active and passive flexion of the knee

Perhaps our most important observation was that passive knee flexion unaccompanied by activation of the TA produces no inhibition of the soleus H-reflex. The inhibition of the soleus H-reflex, when it occurs, is tied to the activation of the TA. Thus to the extent that stretch of the quadriceps is capable of inhibiting the soleus H-reflex, this effect is likely mediated by the classical mechanism(s) of reciprocal inhibition between antagonistic muscles. The neural pathway may involve branches of quadriceps group Ia afferents making synaptic contact with Ia-inhibitory interneurons mediating reciprocal inhibition between the TA and soleus. The existence of this circuit would not be surprising because flexion of the knee and ankle are closely linked, such as during the swing phase of walking or during triple flexion of the leg when moving up steps. Brooke et al. (1995) did report that in one of four subjects tested imposed movements of the leg elicited activity in the TA (50% of its value during stepping). However, they concluded that this was insufficient to account for the inhibition, because the H-reflex was more suppressed during the imposed movements compared with similar levels of tonic TA activity. However, we have shown that reciprocal inhibition of soleus H-reflex is far greater during phasic activity of the TA than during comparable tonic contractions (Lavoie et al. 1997). Furthermore, it is not unreasonable to suggest that Ia-inhibitory interneurons may be more excitable than the larger TA α-motoneurons. Consequently, inhibition of the soleus α-motoneurons may well occur, as detected by H-reflex measurements, without concomitant activity of the TA α-motoneurons. This may well explain why in some subjects no activity in the TA is observed after an imposed flexion of the knee.

When subjects produced a voluntary flexion of the knee, the inhibition of the soleus H-reflex was completed prior to movement about the knee or any other leg joint. This is yet another example showing that the CNS is clearly capable of completely suppressing the H-reflex prior to movement onset, and moreover, that it does not rely on afferent feedback elicited by stretch of the quadriceps to do so.

In summary, the present observations taken together strongly suggest that the input-output parameters of the H-reflex pathway are centrally determined in a task-dependent manner (Capaday and Stein 1986; Lavoie et al. 1997).

General considerations

The idea that the CNS passively allows peripheral inputs to exert powerful effects on central circuits is contrary to the well-established concept of task-dependent control of reflex parameters (e.g., Duysens et al. 1990; Horak 1996; Stein and Capaday 1988). Even if a peripheral influence is shown to have an effect in one task (e.g., imposed joint rotation), it does not follow that the same input will be effective in another task. Thus in relation to the present problem, although it may be that imposed knee flexion can cause inhibition of the H-reflex in passive subjects (we suggest that this is related to activation of the TA), it does not follow that this input will be effective during walking. The suppression of inhibitory effects from the femoral (Faist et al. 1996) and common peroneal nerves (Capaday et al. 1995) on the soleus H-reflex during human walking are clear examples of this point. The inhibition of the soleus H-reflex during the reaction time of a voluntary knee flexion is yet another example. Thus although certain reflex pathways may produce measurable effects in so-called passive conditions during behaviors such as walking or voluntary knee flexion, these circuits are taken over by central control mechanisms. The observation that a cutaneous input from the dorsum of the paw produces very different reflex responses when applied during stance versus swing is a classic example em-

phasizing strong central control of reflex pathways (Forssberg et al. 1977). This observation also demonstrates that even when a peripheral influence is clearly shown to have an effect on the motor output, this is as a result of a permissive action by the CNS.

CONCLUSION

In conclusion, the modulation pattern of the soleus H-reflex during walking follows the classic pattern of centrally produced reciprocal inhibition between antagonistic muscles. All available evidence, including that presented in Fig. 8, show that the CNS adjusts the excitability of the spinal circuits in anticipation of movement-related events rather than as a consequence (see the references in Lavoie et al. 1997). This does not exclude the possibility that peripheral feedback could reinforce the excitability changes produced centrally (e.g., Yang et al. 1991), but movement-related afferent activity is not the primary cause of the time and task-dependent changes observed during human walking.

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